Foley, S.
09/702498

Seq ID 3

FILE 'REGISTRY' ENTERED AT 12:07:31 ON 19 MAR 2003
L1 105281 S C[UT][UT][UT]CACCC/SQSN

FILE 'HCAPLUS' ENTERED AT 12:08:12 ON 19 MAR 2003
L2 5217 S L1
L3 17 S L2 AND (PARAMYXOVIR? OR SENDAI)

L3 ANSWER 1 OF 17 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2003:133305 HCAPLUS
TITLE: Reagents interfering binding interaction between chemokine receptor and surface protein of infectious virus for diagnosis, therapy and screening of antiviral and antiinflammatory agents
INVENTOR(S): Renzi, Paolo; Zemzoumi, Khalid; Lamkhioued, Bouchaid
PATENT ASSIGNEE(S): Topigen Pharmaceutique Inc., USA
SOURCE: PCT Int. Appl., 119 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003014153	A2	20030220	WO 2002-CA1248	20020812

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2001-311088P P 20010810

AB Methods, reagents and compns. for the treatment, prevention and diagnostic of virus infections in vertebrates and more particularly in human and animals are described. The invention provides evidence that the CCR1, CCR2, CCR3, CCR4, CCR5 and CCR8 receptors are involved in human respiratory syncytial virus (RSV) infections. Therefore, the present invention describes methods for modulation of cellular viral infection by modulating a binding interaction between a CCR1, CCR2, CCR3, CCR4, CCR5 and/or CCR8 receptor and a surface protein of the virus. The invention also profits of such a binding interaction for 10 providing methods for reducing viral infection of a cell; methods of attenuating the ability of a pneumovirus to bind a mammalian cell; methods for reducing the initiation or spread of a respiratory tract disease due to human RSV; methods for detecting the presence of a pneumovirus in a biol. sample; gene therapy methods, and methods for identifying novel antiviral and anti-inflammatory 15 compds.

IT INDEXING IN PROGRESS
IT **391849-10-8**, GenBank U95626
RL: ARU (Analytical role, unclassified); BSU (Biological study,

unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(reagents interfering binding interaction between chemokine receptor and surface protein of infectious virus for diagnosis, therapy and screening of antiviral and antiinflammatory agents)

L3 ANSWER 2 OF 17 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:869614 HCAPLUS
DOCUMENT NUMBER: 137:364404
TITLE: Envelope gene-deficient **paramyxovirus**, such as **Sendai** virus, vector for gene therapy
INVENTOR(S): Kitazato, Kaio; Shu, Tsugumine; Kuma, Hidekazu; Ueda, Yasuji; Asakawa, Makoto; Hasegawa, Mamoru; Iida, Akihiro; Tokitou, Fumino; Hirata, Takahiro; Tokusumi, Tsuyoshi; Inoue, Makoto
PATENT ASSIGNEE(S): Japan
SOURCE: U.S. Pat. Appl. Publ., 132 pp., Cont.-in-part of Appl. No. PCT/JP00/03195.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002169306	A1	20021114	US 2001-966277	20010927
WO 2000070070	A1	20001123	WO 2000-JP3195	20000518

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.:
JP 1999-200739 A 19990518
WO 2000-JP3195 A2 20000518
JP 2001-283451 A 20010918

AB Neg.-strand RNA viruses have many advantages as gene introducing vectors. The aim of present invention is to provide a **paramyxovirus** vector deficient in an envelope gene. The virus vector comprises **paramyxovirus**-derived neg.-stranded single-stranded RNA modified not to express at least one envelope protein. To construct a **paramyxovirus** vector suitable for gene therapy, which completely lacks a propagation capability, the present inventors deleted F gene of **Sendai** virus (SeV) from the genome to establish a method to recover infectious virus particles in cells expressing F protein of SeV, using cDNA in which GFP gene is introduced as a reporter. Through this F gene-deficient virus vector, a gene is introduced into rat neuronal cells in primary cultures, primitive mouse blood stem cells, human normal cells, and various other types of cells with a high efficiency, and a high expression was seen. Also, F gene and HN gene-deficient virus virions are successfully recovered by using a virus genomic

cDNA deficient in both F gene and HN gene. Further, F gene and HN gene-deficient infectious viral particles are successfully produced by using F- and HN-expressing cells as helper cells. A virus deficient in F gene and HN gene and having F protein is constructed by using F-expressing cells as helper cells. In addn., M gene-deficient infectious virus particles were produced using helper cells expressing M protein. From cells infected with M gene-deficient viruses, release of virus-like particles was inhibited. Further, a VSV-G pseudotyped virus is successfully constructed by using VSV-G-expressing cells. Techniques for constructing these deficient viruses contribute to the development of vectors of **Paramyxoviridae** usable in gene therapy.

IT **475310-42-0 475310-56-6 475310-58-8**
475310-68-0 475310-76-0 475310-84-0
RL: PRP (Properties)
(unclaimed nucleotide sequence; envelope gene-deficient **paramyxovirus**, such as **Sendai** virus, vector for gene therapy)

L3 ANSWER 3 OF 17 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:721115 HCAPLUS
DOCUMENT NUMBER: 137:258459
TITLE: Positional effect of transgene insertion on expression level in **Paramyxovirus** vectors
INVENTOR(S): Tokusumi, Takeshi; Iida, Akihiro; Hasegawa, Mamoru
PATENT ASSIGNEE(S): Dinabeck Laboratory K. K., Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 27 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 2002272465	A2	20020924	JP 2001-145935	20010516
PRIORITY APPLN. INFO.:			JP 2000-152726 A	20000518
			CA 2000-2322057 A	20001027

AB Virus vectors contg. a transgene placed downstream of viral protein coding genes comprising a **Paramyxovirus**, and use in regulating expression level of transgene, are disclosed. **Sendai** virus (SeV) is an enveloped virus with a nonsegmented neg. strand RNA genome. The recovery of infectious virus from cDNA and generation of recombinant SeV carrying a foreign gene at the promoter proximal position has been demonstrated. In this study, we constructed a series of recombinant SeVs carrying a reporter human secreted alk. phosphatase (SEAP) gene at each viral gene junction or the 5' distal end in order to measure the expression level of the foreign gene. We demonstrated that there was a gradient in the reporter gene expression level that depended on location, due to the polarity of transcription. Insertion of the transgene on the upstream side (3' of - strand), i.e., upstream of NP gene or between NP gene and P gene, was correlated with higher expression level. Transgene insertion on the downstream side (5' of - strand), i.e., downstream of L gene or between HN gene and L gene, on the other hand, was correlated with lower expression level. In contrast, the

growth and final titers of these recombinant viruses showed an opposite gradient to the foreign gene expression level. This suggests the potential for matching therapeutic gene expression level to individual therapy programs by changing the position of the foreign gene when SeVs are used as vectors for human gene therapy.

IT **461480-30-8 461480-45-5 461480-55-7**
461480-63-7 461480-69-3
RL: PRP (Properties)
(unclaimed nucleotide sequence; positional effect of transgene insertion on expression level in **Paramyxovirus** vectors)

L3 ANSWER 4 OF 17 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:408819 HCAPLUS
DOCUMENT NUMBER: 136:396960
TITLE: **Paramyxovirus** vector encoding angiogenesis gene and use for tissue-specific gene transfer and gene therapy
INVENTOR(S): Yonemitsu, Yoshikazu; Sueishi, Katsuo; Fukumura, Masayuki; Hou, Xiaogang; Hasegawa, Mamoru
PATENT ASSIGNEE(S): Dnavec Research Inc., Japan
SOURCE: PCT Int. Appl., 94 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002042481	A1	20020530	WO 2001-JP10323	20011127
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2002024113	A5	20020603	AU 2002-24113	20011127

PRIORITY APPLN. INFO.: JP 2000-359374 A 20001127
WO 2001-JP10323 W 20011127

AB **Paramyxovirus** vector encoding an angiogenesis gene and use for tissue-specific angiogenesis gene transfer and gene therapy for ischemia are disclosed. **Sendai** virus (SeV) lacking the F gene is used. Recent studies suggest the possible therapeutic effect of i.m. vascular endothelial growth factor (VEGF) gene transfer in individuals with crit. limb ischemia. Little information, however, is available regarding (1) the required expression level of VEGF for therapeutic effect, (2) the related expression of endogenous angiogenic factors, including fibroblast growth factor-2 (FGF-2), and (3) the related adverse effects due to overexpression of VEGF. To address these issues, the authors tested effects of overexpression of VEGF165 using recombinant **Sendai** virus (SeV), as directly compared with FGF-2 gene transfer. I.m. injection of SeV strongly boosted FGF-2, resulting

in significant therapeutic effects for limb salvage with increased blood perfusion assocd. with enhanced endogenous VEGF expression in murine models of crit. limb ischemia. In contrast, VEGF165 overexpression, 5-times higher than that of baseline on day 1, also strongly evoked endogenous VEGF in muscles, resulting in an accelerated limb amputation without recovery of blood perfusion. Interestingly, viable skeletal muscles of either VEGF165- or FGF-2-treated ischemic limbs showed similar platelet-endothelial cell adhesion mol.-1-pos. vessel densities. Maturation of newly formed vessels suggested by smooth muscle cell actin-pos. cell lining, however, was significantly disturbed in muscles with VEGF. Further, therapeutic effects of FGF-2 were completely diminished by anti-VEGF neutralizing antibody in vivo, thus indicating that endogenous VEGF does contribute to the effect of FGF-2. These results suggest that VEGF is necessary, but should be delicately regulated to lower expression to treat ischemic limb. The therapeutic effect of FGF-2, assocd. with the harmonized angiogenic effects seen with endogenous VEGF, provides important insights into therapeutic angiogenesis.

IT **429972-58-7 429972-70-3 429972-72-5**
RL: PRP (Properties)
(unclaimed nucleotide sequence; **paramyxovirus** vector encoding angiogenesis gene and use for tissue-specific gene transfer and gene therapy)

REFERENCE COUNT: 10 THERE ARE 10 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 5 OF 17 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:368619 HCAPLUS
DOCUMENT NUMBER: 136:366148
TITLE: **Paramyxovirus** vector for gene transfer to the cardiovascular system
INVENTOR(S): Griesenbach, Uta; Ferrari, Stefano; Geddes, Duncan M.; Alton, Eric W. F. W.; Hasegawa, Mamoru; Hou, Xiaogang
PATENT ASSIGNEE(S): Dnavec Research Inc., Japan
SOURCE: PCT Int. Appl., 67 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002038726	A2	20020516	WO 2001-JP9786	20011108

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

JP 2002142770	A2	20020521	JP 2000-339942		20001108
AU 2002012733	A5	20020521	AU 2002-12733		20011108
PRIORITY APPLN. INFO.:			JP 2000-339942	A	20001108
			WO 2001-JP9786	W	20011108

AB The invention concerns a **paramyxovirus** vector for gene transfer to the cardiovascular system and uses thereof. The invention enables the efficient transfer of a foreign gene product to the cardiovascular system by use of the **paramyxovirus** vector. Products of genes introduced by intranasal or i.m. administration of the **paramyxovirus** vector were detected in blood at high levels. The administration of a vector for the expression of the anti-inflammatory cytokine IL-10 inhibited collagen deposition in lung of pulmonary fibrosis model animal. Thus, the vector of the present invention is suitable for gene transfer to the cardiovascular system.

IT **422351-17-5**
RL: PRP (Properties)
(unclaimed nucleotide sequence; **paramyxovirus** vector for gene transfer to the cardiovascular system)

L3 ANSWER 6 OF 17 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:293834 HCAPLUS
DOCUMENT NUMBER: 136:320347
TITLE: Recombinant **paramyxovirus** vector for skeletal muscle gene transfer
INVENTOR(S): Hukumura, Masayuki; Shiotani, Akihiro; Maeda, Mitsuyo; Hasegawa, Mamoru
PATENT ASSIGNEE(S): Dnavec Research Inc., Japan
SOURCE: PCT Int. Appl., 56 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002031138	A1	20020418	WO 2001-JP8372	20010926
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2001090273	A5	20020422	AU 2001-90273	20010926
PRIORITY APPLN. INFO.:			JP 2000-308533 A	20001006
			WO 2001-JP8372 W	20010926

AB **Paramyxovirus** vectors for high-level expression of a transgene in the skeletal muscle and use in gene therapy of neuromuscular disorders with insulin-like growth factor gene transfection, are disclosed. The authors scrutinized the applicability and efficacy of **Sendai** virus (SeV) vectors expressing either LacZ or human insulin-like growth factor-I

(hIGF-I) in gene transfer into skeletal muscle. Seven days after the i.m. injection of LacZ/SeV X-gal labeled myofibers were demonstrated in rat anterior tibialis muscle with/without bupivacaine treatment and the transgene expression persisted up to 1 mo after injection. Recombinant hIGF-I was detected as a major protein species in culture supernatants of a neonatal rat myoblast cell line L6 and thus induced the cells to undergo myogenetic differentiation. The introduction of hIGF-I/SeV into the muscle showed a significant increase in regenerating and split myofibers which were indicative of hypertrophy, and also an increase in the total no. of myofibers, in comparison to that seen in the LacZ/SeV-treated control muscle. These results demonstrate that SeV achieves high-level transgene expression in skeletal muscle, and that hIGF-I gene transfer using SeV vector may therefore have great potential in the treatment of neuromuscular disorders.

IT **412113-35-0**, 4: PN: WO0231138 SEQID: 4 unclaimed DNA
RL: PRP (Properties)
(unclaimed nucleotide sequence; recombinant **paramyxovirus** vector for skeletal muscle gene transfer)

REFERENCE COUNT: 15 THERE ARE 15 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 7 OF 17 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:90572 HCAPLUS
DOCUMENT NUMBER: 136:139817
TITLE: Negative-sense RNA virus vector for nerve cell targeting
INVENTOR(S): Fukumura, Masayuki; Asakawa, Makoto; Hasegawa, Mamoru; Shirakura, Masayuki
PATENT ASSIGNEE(S): Dnavec Research, Inc., USA
SOURCE: U.S. Pat. Appl. Publ., 25 pp., Cont.-in-part of U.S. Ser. No. 720,979.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002012995	A1	20020131	US 2001-843922	20010430
WO 2000001837	A1	20000113	WO 1999-JP3552	19990701

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: JP 1998-204333 A 19980703
WO 1999-JP3552 W 19990701
US 2001-720979 A2 20010307

AB Use of a neg.-sense RNA virus vector has enabled transfer of nucleic acid into nerve cells. The method of this invention can be used for introducing a gene efficiently into nerve cells including central

nervous system tissue in gene therapy, etc.

IT **391757-57-6**
RL: PRP (Properties)
(unclaimed nucleotide sequence; neg.-sense RNA virus vector for nerve cell targeting)

L3 ANSWER 8 OF 17 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:10315 HCAPLUS
DOCUMENT NUMBER: 136:74656
TITLE: Virus vector for transferring gene into renal cells
INVENTOR(S): Imai, Enyu; Isaka, Yoshitaka; Fukumura, Masayuki; Hasegawa, Mamoru
PATENT ASSIGNEE(S): Dnavec Research Inc., Japan
SOURCE: PCT Int. Appl., 75 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002000264	A1	20020103	WO 2001-JP5513	20010627
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 2001066337	A5	20020108	AU 2001-66337	20010627

PRIORITY APPLN. INFO.: JP 2000-197870 A 20000627
JP 2000-2000197870A 20000627
WO 2001-JP5513 W 20010627

AB Disclosed is a virus vector whereby a gene can be transferred into renal cells at a high efficiency. Use of a **paramyxovirus** vector makes it possible to transfer a gene into renal cells at a high efficiency. The gene transferred by the in vivo administration can be continuously expressed in the renal cells over a prolonged period of time. This vector is adequately usable in gene therapy for kidney. Green fluorescent protein (GFP) gene-encoding recombinant **Sendai** virus vector was prepd. and administered into the renal artery of rats.

IT **383928-77-6**
RL: PRP (Properties)
(unclaimed nucleotide sequence; virus vector for transferring gene into renal cells)

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 9 OF 17 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:730582 HCAPLUS

DOCUMENT NUMBER: 135:287515
TITLE: AIDS virus vaccine with the use of **Sendai** virus vector
INVENTOR(S): Kano, Munehide; Matano, Tetsuro; Kato, Atsushi; Nagai, Yoshiyuki; Hasegawa, Mamoru
PATENT ASSIGNEE(S): Dnavec Research Inc., Japan; Japan as Represented by Director General of National Institute of Infectious Diseases
SOURCE: PCT Int. Appl., 92 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001072340	A1	20011004	WO 2001-JP2769	20010330
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 2002002143	A1	20020103	US 2001-823699	20010330
EP 1270016	A1	20030102	EP 2001-917712	20010330
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				

PRIORITY APPLN. INFO.: US 2000-193127P P 20000330
WO 2001-JP2769 W 20010330

AB A vaccine contg. a **Sendai** virus vector which encodes the viral protein of immunodeficiency virus. Efficient protective immunity can be successfully induced by nasally administering a **Sendai** virus encoding the viral protein of immunodeficiency virus to macaque. By nasally inoculating the vaccine, the expression of an antigen gene mediated by the **Sendai** virus vector is detected in the nasal mucosa and local lymph nodes and an antigen-specific cell-mediated immune response is induced at a significant level. No pathol. sign caused by the vaccination is obsd. When the effect is examd. by exposing the vaccinated animal to Simian immunodeficiency virus, a significant decrease in the plasma virus level is obsd. compared with a control animal. This vaccine is likely useful as an AIDS vaccine.

IT **339958-50-8 364169-71-1**
RL: PRP (Properties)
(unclaimed nucleotide sequence; aIDS virus vaccine with the use of **Sendai** virus vector)

REFERENCE COUNT: 12 THERE ARE 12 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 10 OF 17 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:545865 HCAPLUS
DOCUMENT NUMBER: 135:133090

TITLE: Use of **paramyxovirus** vector in gene transfer into blood vessel
INVENTOR(S): Masaki, Ichirou; Yonemitsu, Yoshikazu; Sueishi, Katsuo; Hasegawa, Mamoru; Kinoh, Hiroaki
PATENT ASSIGNEE(S): Dnavec Research Inc., Japan
SOURCE: PCT Int. Appl., 84 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001053491	A1	20010726	WO 2001-JP87	20010111
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 2001025487	A5	20010731	AU 2001-25487	20010111
EP 1251174	A1	20021023	EP 2001-900664	20010111
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				

PRIORITY APPLN. INFO.: JP 2000-14136 A 20000119
WO 2001-JP87 W 20010111

AB Use of a **paramyxovirus** vector for gene transfer into blood vessels, and kits are disclosed. The gene transfer efficiency into the vascular media layer is significantly elevated by treating the blood vessel with a protease, such as collagenase, urokinase, elastase, tissue plasminogen activator, plasmin, matrix metalloproteinase, to degrade extracellular matrix. The expression of the transgene remains stable over a long time in the vascular cells. Use of this method makes it possible to efficiently transfer a gene within a short period of time into endothelium of the vascular inner cavity, medial layer, outermembrane, etc. Use of **sendai** virus vector for luciferase reporter gene transfer into bovine aortic smooth muscle cells (BSMC), COS-7 cells, and HEK293 cells, is described.

IT **339958-50-8**
RL: PRP (Properties)
(unclaimed nucleotide sequence; use of **paramyxovirus** vector in gene transfer into blood vessel)

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 11 OF 17 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:365881 HCAPLUS
DOCUMENT NUMBER: 134:362206
TITLE: Systemic autoimmune disease gene therapy with extracellular-superoxide dismutase

INVENTOR(S): Niitsu, Yoshiro; Yamauchi, Takafumi; Iyama, Satoshi; Fukumura, Masayuki; Hasegawa, Mamoru
PATENT ASSIGNEE(S): Dinabeck Laboratory K. K., Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 29 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 2001136985	A2	20010522	JP 2000-264304	20000831

PRIORITY APPLN. INFO.: JP 1999-248032 A 19990901

AB Gene therapy for systemic autoimmune diseases with extracellular-superoxide dismutase (EC-SOD) gene is disclosed. Delivery of the human EC-SOD gene by retrovirus vector or **Sendai** virus vector, and cells transformed with the vector, are claimed. Treatment of chronic rheumatoid arthritis or colitis is claimed. Extracellular-superoxide dismutase [EC 1.15.1.1] (EC-SOD) is a secretory glycoprotein with high affinity for heparin. This enzyme locates in blood vessel walls at a high enough level to suppress oxidative stress under normal conditions. EC-SOD is the major SOD isoenzyme in plasma, anchored to heparan sulfate proteoglycans in the glycocalyx of endothelial cell surfaces. Introduction of human EC-SOD gene into mouse fibroblast and obsd. suppression of collagen-induced arthritis and colitis are described.

IT **339958-50-8 340053-06-7**
RL: PRP (Properties)
(unclaimed nucleotide sequence; systemic autoimmune disease gene therapy with extracellular-superoxide dismutase)

L3 ANSWER 12 OF 17 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:338737 HCAPLUS
DOCUMENT NUMBER: 134:351843
TITLE: Recombinant **sendai** virus vector for introducing exogenous genes to airway epithelia and gene therapy of cystic fibrosis
INVENTOR(S): Yonemitsu, Yoshikazu; Hasegawa, Mamoru; Alton, Eric
PATENT ASSIGNEE(S): Dnavec Research Inc., Japan
SOURCE: PCT Int. Appl., 42 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001032898	A2	20010510	WO 2000-JP7737	20001102
WO 2001032898	A3	20010907		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ,

TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

EP 1228232 A2 20020807 EP 2000-971756 20001102
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR

PRIORITY APPLN. INFO.: US 1999-163055P P 19991102
JP 1999-359218 A 19991217
WO 2000-JP7737 W 20001102

AB Provided are a recombinant **Sendai** virus vector for introducing exogenous genes to airway epithelia and a method for introducing exogenous genes using the vector. The recombinant **Sendai** virus vector enables efficient gene transfer to native mucus-layered airway epithelial cells by briefly contacting the vector with the cells. Furthermore, the vector can introduce genes to not only apical surfaces but also submucosal glands where CFTR primarily expresses. The vector can thus be used for gene therapy of CF, a CFTR-deficient disease.

IT **339040-33-4**, 5: PN: WO0132898 SEQID: 4 unclaimed DNA
339958-50-8
RL: PRP (Properties)
(unclaimed nucleotide sequence; recombinant **sendai** virus vector for introducing exogenous genes to airway epithelia and gene therapy of cystic fibrosis)

IT **339040-34-5**
RL: PRP (Properties)
(unclaimed sequence; recombinant **sendai** virus vector for introducing exogenous genes to airway epithelia and gene therapy of cystic fibrosis)

L3 ANSWER 13 OF 17 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2000:824432 HCAPLUS
DOCUMENT NUMBER: 134:15152
TITLE: **Paramyxoviridae** virus vector defective in envelope gene F and its construction
INVENTOR(S): Li, Hai-Ou; Shu, Tsugumine; Kuma, Hidekazu; Ueda, Yasuji; Asakawa, Makoto; Hasegawa, Mamoru; Iida, Akihiro; Tokitou, Fumino; Hirata, Takahiro; Tokusumi, Tsuyoshi
PATENT ASSIGNEE(S): DNAVEC Research Inc., Japan
SOURCE: PCT Int. Appl., 177 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000070070	A1	20001123	WO 2000-JP3195	20000518

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

EP 1186667 A1 20020313 EP 2000-927807 20000518

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO

US 2002169306 A1 20021114 US 2001-966277 20010927

PRIORITY APPLN. INFO.: JP 1999-200739 A 19990518
WO 2000-JP3195 W 20000518
JP 2001-283451 A 20010918

AB Virus virions defective in F gene are successfully collected by using a **Sendai** virus genomic cDNA with deletion of F gene. Further, infectious viral particles defective in F gene are successfully constructed by using F-expression cells as helper cells. Also, virus virions defective in F gene and HN gene are successfully collected by using a virus genomic cDNA with deletion of both of F gene and HN gene. Further, infectious viral particles defective in F gene and HN gene are successfully produced by using F- and HN-expression cells as helper cells. A virus being defective in F gene and HN gene and having F protein is constructed by using F-expression cells as helper cells. Further, a VSV-G pseudo type virus is successfully constructed by using VSV-G-expression cells. Techniques for constructing these defective viruses contribute to the development of vectors of **Paramyxoviridae** usable in gene therapy.

IT **308388-99-0 308389-13-1 308389-15-3 308389-25-5 308389-33-5**
RL: PRP (Properties)
(unclaimed sequence; **paramyxoviridae** virus vector defective in envelope gene F and its construction)

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 14 OF 17 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2000:824418 HCAPLUS
DOCUMENT NUMBER: 134:2564
TITLE: RNP originating in **paramyxovirus** and use for preparation of safe viral vectors
INVENTOR(S): Li, Hai-Ou; Shu, Tsugumine; Kuma, Hidekazu; Ueda, Yasuji; Asakawa, Makoto; Hasegawa, Mamoru; Iida, Akihiro; Hirata, Takahiro
PATENT ASSIGNEE(S): DNAVEC Research Inc., Japan
SOURCE: PCT Int. Appl., 172 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000070055	A1	20001123	WO 2000-JP3194	20000518

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG,

US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
EP 1179594 A1 20020213 EP 2000-929790 20000518
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO
US 2003022376 A1 20030130 US 2001-966930 20010927
PRIORITY APPLN. INFO.: JP 1999-200740 A 19990518
WO 2000-JP3194 W 20000518
JP 2001-283451 A 20010918

AB A functional RNP (ribonucleoprotein complex) contg. (-)-chain single stranded RNA originating in **Sendai** virus which has been modified so as not to express any envelope protein. An RNP contg. this foreign gene is prepd. and inserted into a cell with the use of a cationic liposome, thereby successfully expressing the foreign gene. Vectors derived from the NS-RNA (minus-stranded RNA) virus are deemed safe for use.

IT **308388-99-0 308389-13-1 308389-15-3 308389-25-5 308389-33-5**
RL: PRP (Properties)
(unclaimed nucleotide sequence; rNP originating in **paramyxovirus** and use for prepn. of safe viral vectors)

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 15 OF 17 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2000:654258 HCAPLUS
DOCUMENT NUMBER: 133:236811
TITLE: **Sendai** virus vector expressing influenza virus hemagglutinin for vaccine use
INVENTOR(S): Kato, Atsushi; Kiyotani, Katsuhiro; Yoshida, Tetsuya; Shiota, Tatsuo; Toriyoshi, Hidenobu; Takebe, Yutaka; Nagai, Yoshiyuki
PATENT ASSIGNEE(S): Dinabeck Laboratory K. K., Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 28 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 2000253876	A2	20000919	JP 1999-60918	19990308

PRIORITY APPLN. INFO.: JP 1999-60918 19990308

AB **Sendai** virus vector capable of expressing influenza virus protein is disclosed. Influenza vaccine based on the vector expressing hemagglutinin (HA) of virulent strain of influenza virus, subtype H5 or H7, and a method of its prodn. are provided. Influenza vaccination method, antigenic protein, immunol. anal. protein, and reagent kits for ELISA are also provided. A recombinant **Sendai** virus vector for expression of influenza virus subtype H5 strain HA gene was constructed. Immunol. anal. of the cells infected by the virus vector confirmed the expression of HA protein. Effectiveness of the vaccine against the influenza virus challenge was demonstrated in mice immunized with

the vaccine. In addn., recombinant **Sendai** virus expressing HIV-1 subtype E gp120 was constructed.

IT **292885-72-4 292885-75-7**
RL: PRP (Properties)
(unclaimed nucleotide sequence; **sendai** virus vector expressing influenza virus hemagglutinin for vaccine use)

L3 ANSWER 16 OF 17 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2000:441388 HCAPLUS
DOCUMENT NUMBER: 133:72923
TITLE: Preparation of cytokines using a **Sendai** virus expression system
INVENTOR(S): Kai, Chieko; Kato, Atsushi
PATENT ASSIGNEE(S): Nippon Biocapital Limited, Japan
SOURCE: Eur. Pat. Appl., 13 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 1013667	A2	20000628	EP 1999-402786	19991109
EP 1013667	A3	20000705		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
JP 2000201689	A2	20000725	JP 1999-318550	19991109
US 6514728	B1	20030204	US 1999-436504	19991109

PRIORITY APPLN. INFO.: JP 1998-317321 A 19981109

AB The present invention provides a process for prepg. a cytokine inexpensively in large amts. by expressing the cytokine in a hen's egg using **Sendai** virus vector. The cytokine obtained by the present process is expected to be useful as medication because it has sugar chains very similar to those of mammals.

IT **278811-35-1**, 4: PN: EP1013667 SEQID: 4 unclaimed DNA
RL: PRP (Properties)
(unclaimed nucleotide sequence; prepn. of cytokines using a **Sendai** virus expression system)

L3 ANSWER 17 OF 17 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1996:751520 HCAPLUS
DOCUMENT NUMBER: 126:15522
TITLE: Recombinant infectious laryngotracheitis viruses with deletions in genes associated with virulence and their uses as poultry vaccines
INVENTOR(S): Wild, Martha A.; Cochran, Mark D.
PATENT ASSIGNEE(S): Syntro Corporation, USA
SOURCE: PCT Int. Appl., 218 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9629396	A1	19960926	WO 1996-US3916	19960321

W: AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, GB, GE, HU, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML

CA 2216139	AA	19960926	CA 1996-2216139	19960321
AU 9653690	A1	19961008	AU 1996-53690	19960321
AU 721451	B2	20000706		
EP 822980	A1	19980211	EP 1996-910515	19960321
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 11503009	T2	19990323	JP 1996-528630	19960321

PRIORITY APPLN. INFO.: US 1995-410121 A 19950323
US 1995-468190 A1 19950606
WO 1996-US3916 W 19960321

AB The present invention provides a recombinant, attenuated infectious laryngotracheitis laryngotracheitis virus (ILTV) genome which contains a deletion in the unique short region of the viral genomic DNA. Thus, an 18,912-nucleotide region was sequenced which contains the unique short region and its terminal repeats; a genomic map and organization of the genes within this region were detd. This region contains genes that are assocd. with ILTV virulence and a deletion in those genes leads to an attenuated ILTV which is useful as a vaccine against subsequent attack by a virulent ILTV strain. The recombinant, attenuated infectious laryngotracheitis virus comprising the infectious laryngotracheitis viral genome which can contain a deletion in the glycoprotein gG gene, US2 gene, UL47-like gene, ORF4 gene, or glycoprotein g60 gene. Thus, S-ILT-015 is an infectious laryngotracheitis virus that has an .apprx.2640-bp deletion of the UL47-like gene, the ORF4 gene, and the gG gene. The gene for Escherichia coli .beta.-glucuronidase (uidA) was inserted in the place of the UL47-like, ORF4, and gG genes and is under the control of the pseudorabies virus gX promoter. S-ILT-015 is useful as an attenuated live vaccine or as a killed vaccine to protect chickens from ILT disease. The present invention also provides a method for distinguishing chickens or other poultry vaccinated with a recombinant infectious laryngotracheitis virus which produces no glycoprotein gG from those infected with a naturally-occurring infectious laryngotracheitis virus. Antigens from various disease-causing microorganisms (e.g., the spike and matrix protein genes from infectious bronchitis virus) can be inserted into the recombinant, attenuated ILTV to induce protective antibodies to avian disease.

IT **179907-86-9 183748-82-5**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(nucleotide sequence; recombinant infectious laryngotracheitis viruses with deletions in genes assocd. with virulence and their uses as poultry vaccines)

IT **183748-98-3**
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(nucleotide sequence; recombinant infectious laryngotracheitis viruses with deletions in genes assocd. with virulence and their uses as poultry vaccines)

E1 THROUGH E35 ASSIGNED

FILE 'REGISTRY' ENTERED AT 12:11:14 ON 19 MAR 2003
L4 35 SEA FILE=REGISTRY ABB=ON PLU=ON (339958-50-8/BI OR
308388-99-0/BI OR 308389-13-1/BI OR 308389-15-3/BI OR
308389-25-5/BI OR 308389-33-5/BI OR 179907-86-9/BI OR
183748-82-5/BI OR 183748-98-3/BI OR 278811-35-1/BI OR
292885-72-4/BI OR 292885-75-7/BI OR 339040-33-4/BI OR
339040-34-5/BI OR 340053-06-7/BI OR 364169-71-1/BI OR
383928-77-6/BI OR 391757-57-6/BI OR 391849-10-8/BI OR
412113-35-0/BI OR 422351-17-5/BI OR 429972-58-7/BI OR
429972-70-3/BI OR 429972-72-5/BI OR 461480-30-8/BI OR
461480-45-5/BI OR 461480-55-7/BI OR 461480-63-7/BI OR
461480-69-3/BI OR 475310-42-0/BI OR 475310-56-6/BI OR
475310-58-8/BI OR 475310-68-0/BI OR 475310-76-0/BI OR
475310-84-0/BI)

L4 ANSWER 1 OF 35 REGISTRY COPYRIGHT 2003 ACS
RN **475310-84-0** REGISTRY
CN DNA, d(C-T-T-T-C-A-C-C-C-T) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 65: PN: US20020169306 SEQID: 63 unclaimed DNA
SQL 10
MF Unspecified
CI MAN

REFERENCE 1: 137:364404

L4 ANSWER 2 OF 35 REGISTRY COPYRIGHT 2003 ACS
RN **475310-76-0** REGISTRY
CN 52: PN: US20020169306 SEQID: 52 unclaimed DNA (9CI) (CA INDEX NAME)
SQL 72
MF Unspecified
CI MAN

REFERENCE 1: 137:364404

L4 ANSWER 3 OF 35 REGISTRY COPYRIGHT 2003 ACS
RN **475310-68-0** REGISTRY
CN DNA, d(G-A-T-A-T-C-T-G-C-C-A-T-T-G-C-G-C-C-G-T-G-C-T-A-G-C-T-G-A-A-A-
T-T-T-C-T-T-T-C-A-C-C-C-T-A-A-G) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 44: PN: US20020169306 SEQID: 44 unclaimed DNA
SQL 47
MF Unspecified
CI MAN

REFERENCE 1: 137:364404

L4 ANSWER 4 OF 35 REGISTRY COPYRIGHT 2003 ACS
RN **475310-58-8** REGISTRY
CN 34: PN: US20020169306 SEQID: 34 unclaimed DNA (9CI) (CA INDEX NAME)
SQL 72
MF Unspecified
CI MAN

REFERENCE 1: 137:364404

L4 ANSWER 5 OF 35 REGISTRY COPYRIGHT 2003 ACS
RN **475310-56-6** REGISTRY
CN 32: PN: US20020169306 SEQID: 32 unclaimed DNA (9CI) (CA INDEX NAME)
SQL 74
MF Unspecified
CI MAN

REFERENCE 1: 137:364404

L4 ANSWER 6 OF 35 REGISTRY COPYRIGHT 2003 ACS
RN **475310-42-0** REGISTRY
CN 18: PN: US20020169306 SEQID: 18 unclaimed DNA (9CI) (CA INDEX NAME)
SQL 80
MF Unspecified
CI MAN

REFERENCE 1: 137:364404

L4 ANSWER 7 OF 35 REGISTRY COPYRIGHT 2003 ACS
RN **461480-69-3** REGISTRY
CN 40: PN: JP2002272465 SEQID: 40 unclaimed DNA (9CI) (CA INDEX NAME)
SQL 60
MF Unspecified
CI MAN

REFERENCE 1: 137:258459

L4 ANSWER 8 OF 35 REGISTRY COPYRIGHT 2003 ACS
RN **461480-63-7** REGISTRY
CN 34: PN: JP2002272465 SEQID: 34 unclaimed DNA (9CI) (CA INDEX NAME)
SQL 72
MF Unspecified
CI MAN

REFERENCE 1: 137:258459

L4 ANSWER 9 OF 35 REGISTRY COPYRIGHT 2003 ACS
RN **461480-55-7** REGISTRY
CN DNA, d(G-A-T-A-T-C-T-G-C-C-A-T-T-G-C-G-C-C-G-T-G-C-T-A-G-C-T-G-A-A-A-T-T-T-C-T-T-T-C-A-C-C-C-T-A-A-G) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 26: PN: JP2002272465 SEQID: 26 unclaimed DNA
SQL 47
MF Unspecified
CI MAN

REFERENCE 1: 137:258459

L4 ANSWER 10 OF 35 REGISTRY COPYRIGHT 2003 ACS
RN **461480-45-5** REGISTRY
CN 16: PN: JP2002272465 SEQID: 16 unclaimed DNA (9CI) (CA INDEX NAME)
SQL 74
MF Unspecified
CI MAN

REFERENCE 1: 137:258459

L4 ANSWER 11 OF 35 REGISTRY COPYRIGHT 2003 ACS
RN **461480-30-8** REGISTRY
CN DNA, d(C-T-T-T-C-A-C-C-C-T) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 1: PN: JP2002272465 SEQID: 1 unclaimed DNA
SQL 10
MF Unspecified
CI MAN

REFERENCE 1: 137:258459

L4 ANSWER 12 OF 35 REGISTRY COPYRIGHT 2003 ACS
RN **429972-72-5** REGISTRY
CN 16: PN: WO0242481 SEQID: 16 unclaimed DNA (9CI) (CA INDEX NAME)
SQL 103
MF Unspecified
CI MAN

REFERENCE 1: 136:396960

L4 ANSWER 13 OF 35 REGISTRY COPYRIGHT 2003 ACS
RN **429972-70-3** REGISTRY
CN 14: PN: WO0242481 SEQID: 14 unclaimed DNA (9CI) (CA INDEX NAME)
SQL 82
MF Unspecified
CI MAN

REFERENCE 1: 136:396960

L4 ANSWER 14 OF 35 REGISTRY COPYRIGHT 2003 ACS
RN **429972-58-7** REGISTRY
CN DNA, d(C-T-T-T-C-A-C-C-C-T) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 1: PN: WO0242481 SEQID: 1 unclaimed DNA
SQL 10
MF Unspecified
CI MAN

REFERENCE 1: 136:396960

L4 ANSWER 15 OF 35 REGISTRY COPYRIGHT 2003 ACS
RN **422351-17-5** REGISTRY
CN DNA, d(C-T-T-T-C-A-C-C-C-T) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 1: PN: WO0238726 SEQID: 1 unclaimed DNA
SQL 10
MF Unspecified
CI MAN

REFERENCE 1: 136:366148

L4 ANSWER 16 OF 35 REGISTRY COPYRIGHT 2003 ACS
RN **412113-35-0** REGISTRY
CN 4: PN: WO0231138 SEQID: 4 unclaimed DNA (9CI) (CA INDEX NAME)
SQL 73
MF Unspecified
CI MAN

REFERENCE 1: 136:320347

L4 ANSWER 17 OF 35 REGISTRY COPYRIGHT 2003 ACS
RN **391849-10-8** REGISTRY
CN DNA (human clone BAC 110P12) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 1609: PN: WO0224956 FIGURE: 9 claimed DNA
CN 2312: PN: WO0224956 FIGURE: 13 claimed DNA
CN 548: PN: WO0224956 FIGURE: 1 claimed DNA
CN DNA (human clone BAC 110P12 gene ccr2 plus gene ccr2 plus gene ccr5 plus gene ccr6 plus gene lactoferrin)
CN GenBank U95626
SQL 143068
MF Unspecified
CI MAN

REFERENCE 1: 138:168793

REFERENCE 2: 137:227709

REFERENCE 3: 137:104829

REFERENCE 4: 137:61578

REFERENCE 5: 137:59899

REFERENCE 6: 137:45439

REFERENCE 7: 136:277466

L4 ANSWER 18 OF 35 REGISTRY COPYRIGHT 2003 ACS
RN **391757-57-6** REGISTRY
CN 3: PN: US20020012995 SEQID: 3 unclaimed DNA (9CI) (CA INDEX NAME)
SQL 72
MF Unspecified
CI MAN

REFERENCE 1: 136:139817

L4 ANSWER 19 OF 35 REGISTRY COPYRIGHT 2003 ACS
RN **383928-77-6** REGISTRY
CN DNA, d(C-T-T-T-C-A-C-C-C-T) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 1: PN: WO0200264 SEQID: 1 unclaimed DNA
SQL 10
MF Unspecified
CI MAN

REFERENCE 1: 136:74656

L4 ANSWER 20 OF 35 REGISTRY COPYRIGHT 2003 ACS
RN **364169-71-1** REGISTRY
CN 10: PN: WO0172340 SEQID: 10 unclaimed DNA (9CI) (CA INDEX NAME)
SQL 63
MF Unspecified
CI MAN

REFERENCE 1: 135:287515

L4 ANSWER 21 OF 35 REGISTRY COPYRIGHT 2003 ACS
RN **340053-06-7** REGISTRY
CN 11: PN: JP2001136985 SEQID: 11 unclaimed DNA (9CI) (CA INDEX NAME)
SQL 78
MF Unspecified
CI MAN

REFERENCE 1: 134:362206

L4 ANSWER 22 OF 35 REGISTRY COPYRIGHT 2003 ACS
RN **339958-50-8** REGISTRY
CN DNA, d(C-T-T-T-C-A-C-C-C-T) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 1: PN: WO0153491 SEQID: 1 unclaimed DNA
CN 1: PN: WO0172340 SEQID: 1 unclaimed DNA
CN 2: PN: WO0132898 SEQID: 1 unclaimed DNA
CN 5: PN: JP2001136985 SEQID: 5 unclaimed DNA
SQL 10
MF Unspecified
CI MAN

REFERENCE 1: 135:287515

REFERENCE 2: 135:133090

REFERENCE 3: 134:362206

REFERENCE 4: 134:351843

L4 ANSWER 23 OF 35 REGISTRY COPYRIGHT 2003 ACS
RN **339040-34-5** REGISTRY
CN 1: PN: WO0132898 SEQID: 5 unclaimed sequence (9CI) (CA INDEX NAME)
SQL 76
MF Unspecified
CI MAN

REFERENCE 1: 134:351843

L4 ANSWER 24 OF 35 REGISTRY COPYRIGHT 2003 ACS
RN **339040-33-4** REGISTRY
CN 5: PN: WO0132898 SEQID: 4 unclaimed DNA (9CI) (CA INDEX NAME)
SQL 127
MF Unspecified
CI MAN

REFERENCE 1: 134:351843

L4 ANSWER 25 OF 35 REGISTRY COPYRIGHT 2003 ACS
RN **308389-33-5** REGISTRY
CN 56: PN: WO0070055 SEQID: 52 unclaimed DNA (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 52: PN: WO0070070 SEQID: 52 unclaimed sequence
SQL 72
MF Unspecified
CI MAN

REFERENCE 1: 134:15152

REFERENCE 2: 134:2564

L4 ANSWER 26 OF 35 REGISTRY COPYRIGHT 2003 ACS
RN **308389-25-5** REGISTRY
CN 48: PN: WO0070055 SEQID: 44 unclaimed DNA (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 44: PN: WO0070070 SEQID: 44 unclaimed sequence
SQL 47
MF Unspecified
CI MAN

REFERENCE 1: 134:15152

REFERENCE 2: 134:2564

L4 ANSWER 27 OF 35 REGISTRY COPYRIGHT 2003 ACS
RN **308389-15-3** REGISTRY
CN 37: PN: WO0070055 SEQID: 34 unclaimed DNA (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 34: PN: WO0070070 SEQID: 34 unclaimed sequence
SQL 72
MF Unspecified
CI MAN

REFERENCE 1: 134:15152

REFERENCE 2: 134:2564

L4 ANSWER 28 OF 35 REGISTRY COPYRIGHT 2003 ACS
RN **308389-13-1** REGISTRY
CN 35: PN: WO0070055 SEQID: 32 unclaimed DNA (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 32: PN: WO0070070 SEQID: 32 unclaimed sequence
SQL 74
MF Unspecified
CI MAN

REFERENCE 1: 134:15152

REFERENCE 2: 134:2564

L4 ANSWER 29 OF 35 REGISTRY COPYRIGHT 2003 ACS
RN **308388-99-0** REGISTRY
CN 21: PN: WO0070055 SEQID: 18 unclaimed DNA (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 18: PN: WO0070070 SEQID: 18 unclaimed sequence
SQL 80
MF Unspecified
CI MAN

REFERENCE 1: 134:15152

REFERENCE 2: 134:2564

L4 ANSWER 30 OF 35 REGISTRY COPYRIGHT 2003 ACS
RN **292885-75-7** REGISTRY
CN 5: PN: JP2000253876 SEQID: 5 unclaimed DNA (9CI) (CA INDEX NAME)

SQL 69
MF Unspecified
CI MAN

REFERENCE 1: 133:236811

L4 ANSWER 31 OF 35 REGISTRY COPYRIGHT 2003 ACS
RN **292885-72-4** REGISTRY
CN 2: PN: JP2000253876 SEQID: 2 unclaimed DNA (9CI) (CA INDEX NAME)
SQL 70
MF Unspecified
CI MAN

REFERENCE 1: 133:236811

L4 ANSWER 32 OF 35 REGISTRY COPYRIGHT 2003 ACS
RN **278811-35-1** REGISTRY
CN 4: PN: EP1013667 SEQID: 4 unclaimed DNA (9CI) (CA INDEX NAME)
SQL 58
MF Unspecified
CI MAN

REFERENCE 1: 133:72923

L4 ANSWER 33 OF 35 REGISTRY COPYRIGHT 2003 ACS
RN **183748-98-3** REGISTRY
CN DNA (Gallid herpesvirus 1 strain USDA 83-2 glycoprotein g60 gene) (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Deoxyribonucleic acid (infectious laryngotracheitis virus strain USDA 83-2 glycoprotein g60 gene)
SQL 2958
MF Unspecified
CI MAN

REFERENCE 1: 126:15522

L4 ANSWER 34 OF 35 REGISTRY COPYRIGHT 2003 ACS
RN **183748-82-5** REGISTRY
CN DNA (Gallid herpesvirus 1 strain USDA 83-2 unique short region-containing 13,473-nucleotide fragment) (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Deoxyribonucleic acid (infectious laryngotracheitis virus strain USDA 83-2 unique short region-containing 13,473-nucleotide fragment)
SQL 13473
MF Unspecified
CI MAN

REFERENCE 1: 126:15522

L4 ANSWER 35 OF 35 REGISTRY COPYRIGHT 2003 ACS
RN **179907-86-9** REGISTRY
CN DNA (laryngotracheitis virus strain USDAchallenge gene US10 plus gene US2 plus protein kinase gene plus gene UL47 plus glycoprotein G gene plus glycoprotein D gene plus glycoprotein I gene plus glycoprotein E gene plus flanks) (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Deoxyribonucleic acid (laryngotracheitis virus strain USDAchallenge

gene US10 plus gene US2 plus protein kinase gene plus gene UL47 plus glycoprotein G gene plus glycoprotein D gene plus glycoprotein I gene plus glycoprotein E gene plus 5'- and 3'-flanking region fragment)
OTHER NAMES:
CN Deoxyribonucleic acid (infectious laryngotracheitis virus strain USDA 83-2 unique short region-containing 18,912-nucleotide fragment)
SQL 18912
MF Unspecified
CI MAN

REFERENCE 1: 126:15522

REFERENCE 2: 125:213766

FILE 'HCAPLUS' ENTERED AT 12:12:07 ON 19 MAR 2003
L5 22 S E(W)I(W)S
L6 0 S L5 AND (PARAMYXOVIR? OR SENDAI)
L7 1 S L5 AND SEQUENC?
L8 1 S L7 NOT L3

L8 ANSWER 1 OF 1 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1995:303423 HCAPLUS
DOCUMENT NUMBER: 122:75986
TITLE: A test of lattice protein folding algorithms
AUTHOR(S): Yue, Kaizhi; Fiebig, Klaus M.; Thomas, Paul D.; Chan, Hue Sun; Shakhnovich, Eugene I.; Dill, Ken A.
CORPORATE SOURCE: Department Pharmaceutical Chemistry, University California, San Francisco, CA, 94143-1204, USA
SOURCE: Proceedings of the National Academy of Sciences of the United States of America (1995), 92(1), 325-9
CODEN: PNASA6; ISSN: 0027-8424
PUBLISHER: National Academy of Sciences
DOCUMENT TYPE: Journal
LANGUAGE: English
AB We report a blind test of lattice-model-based search strategies for finding global min. of model protein chains. One of us (**E.I.S.**) selected 10 compact conformations of 48-mer chains on the three-dimensional cubic lattice and used their inverse folding algorithm to design HP (H, hydrophobic; P, polar) **sequences** that should fold to those "target" structures. The **sequences**, but not the structures, were sent to the UCSF group (K.Y., K.M.F., P.D.T., H.S.C., and K.A.D.), who used two methods to attempt to find the globally optimal conformations: "hydrophobic zippers" and a constraint-based hydrophobic core construction (CHCC) method. The CHCC method found global min. in all cases, and the hydrophobic zippers method found global min. in some cases, in minutes to hours on workstations. In 9 out of 10 **sequences**, the CHCC method found lower energy conformations than the 48-mers were designed to fold to. Thus the search strategies succeed for the HP model but the design strategy dose not. For every **sequence** the global energy min. was found to have multiple degeneracy with 103 to 106 conformations. We discuss the implications of these results for (i) searching conformational spaces of simple models of proteins and (ii) how these simple models relate to proteins.

(FILE 'MEDLINE, BIOSIS, EMBASE, WPIDS, CONFSCI, SCISEARCH, JICST-EPLUS, JAPIO' ENTERED AT 12:14:10 ON 19 MAR 2003)
L9 0 S L6
L10 14 S L7
L11 11 DUP REM L10 (3 DUPLICATES REMOVED)

L11 ANSWER 1 OF 11 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2002-519316 [55] WPIDS
DOC. NO. CPI: C2002-146936
TITLE: Rapid determination of protease cleavage site motifs using a mixture-based oriented peptide library.
DERWENT CLASS: B04 D16
INVENTOR(S): CANTLEY, L C; TURK, B E
PATENT ASSIGNEE(S): (BETH-N) BETH ISRAEL DEACONESS MEDICAL CENT
COUNTRY COUNT: 98
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2002038796	A2	20020516	(200255)*	EN	126
AU 2002030630	A	20020521	(200260)		

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2002038796	A2	WO 2001-US46777	20011108
AU 2002030630	A	AU 2002-30630	20011108

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2002030630	A	Based on	WO 200238796

PRIORITY APPLN. INFO: US 2000-246815P 20001108
AN 2002-519316 [55] WPIDS
AB WO 200238796 A UPAB: 20020829
NOVELTY - Determining (M1) an amino acid **sequence** (AAS) motif for a cleavage site (CS) of protease (I), comprising contacting (I) with peptide library containing degenerate residues which allow for cleavage of substrate by (I), allowing (I) to cleave peptides within the degenerate peptide library having CS for (I) to form a population of cleaved peptides, and determining AAS motif for CS of (I), is new.
DETAILED DESCRIPTION - Determining (M1) an amino acid **sequence** (AAS) motif for a cleavage site (CS) of protease (I), comprising:
(a) contacting (I) with a peptide library containing one or

more degenerate residues under conditions which allow for cleavage of a substrate by (I);

(b) allowing (I) to cleave peptides within the degenerate peptide library having a CS for (I) to form a population of cleaved peptides comprising amino-terminal peptides and carboxy terminal peptides;

(c) determining AAS of the population of cleaved carboxy terminal peptides; and

(d) determining AAS motif for CS of (I) based upon the relative abundance of different amino acid residues at each degenerate position within the population of cleaved C-terminal peptides.

INDEPENDENT CLAIMS are also included for the following:

(1) a protease inhibitor (PI1) or substrate comprising a **sequence** determined by M1;

(2) an inhibitor (II) of matrix metalloproteinase protease (MMP) activity comprising a noncleavable peptide molecule comprising **sequence** (S8)-(S13) or its fragment that inhibits MMP activity;

(3) an inhibitor (III) of MMP activity that competes for binding to (II);

(4) a composition (C1) comprising (II) or (III);

(5) an inhibitor (IV) of Bacillus anthracis lethal factor protease activity comprising a noncleavable peptide molecule having a **sequence** of (S2) or its fragment that inhibits lethal factor protease activity;

(6) an inhibitor (V) of B. anthracis lethal factor protease activity comprising any of (S3)-(S7);

(7) an inhibitor (VI) of B. anthracis lethal factor that competes for binding to lethal factor with the inhibitor of (IV) or (V);

(8) a composition (C2) comprising (IV), (V) or (VI);

(9) determining (M2) AAS motif for a binding site of (I), comprising:

(a) contacting (I) with an oriented peptide library containing one or more degenerate residues under conditions which allow for binding of a substrate by (I);

(b) allowing (I) to bind peptides within the degenerate peptide library having a binding site for (I) to form protease-peptide complexes;

(c) isolating the protease-peptide complexes from the unbound peptides;

(d) releasing the peptides from the protease-peptide complexes;

(e) isolating the peptides previously bound to (I);

(f) determining the amino acid **sequences** of the peptides; and

(g) determining AAS motif for a binding site of (I) based upon the relative abundance of different amino acid residues at each degenerate position within the peptides;

(10) a protein binding molecule comprising a **sequence** determined by M2;

(11) an intramolecularly-quenched fluorogenic peptide protease substrate (IFP1) comprising a lethal factor protease cleavage motif **sequence** flanked by a fluorescent group and a fluorescence quenching moiety;

(12) an intramolecularly-quenched fluorogenic peptide protease substrate comprising a lethal factor protease cleavage motif **sequence** flanked by a fluorescent proteins that have overlapping emission spectra;

(13) an intramolecularly-quenched fluorogenic protease substrate comprising a matrix metalloprotease cleavage motif **sequence** flanked by fluorescent proteins that have overlapping emission spectra;

(14) identifying (M3) protease inhibitors, comprising providing a protease and cleavable protease substrate, where the uncleaved substrate is distinguishable from the cleaved substrate;

(15) a protease inhibitor (PI2) identified by M3;

(16) identifying (M4) protease inhibitors, comprising providing a protease, where a protease inhibitor binds the protease, and a candidate protease inhibitor compound; and

(17) a protease inhibitor (PI3) identified by M4.

XaaXaaXaaXaaXaaPXaaPXaaXaa (S2); 2-thioacetyl-Tyr-Pro-Met-amide (S3); alpha -acetyl-Lys-Val-Tyr-Pro-hydroxamic acid (S4); alpha -acetyl-Lys-Val-Tyr- beta Ala-hydroxamic acid (S5); alpha -acetyl-Lys-Pro-Thr-Pro-hydroxamic acid (S6); KKKPYPXaaXaaXaaXaaGK (S7); Xaa1Xaa2Xaa3Xaa4Xaa5Xaa6Xaa7Xaa8 (S8); Xaa9-Val-Pro-Xaa10Xaa11Xaa12 Xaa13Xaa14 (S9); Xaa15Xaa16Xaa17Xaa18Xaa19Xaa20Xaa21 Xaa22 (S10); Val-Val-Xaa23Xaa24-Ser-Xaa25Xaa26Xaa27 (S11); Xaa28Xaa29Xaa30Xaa31Xaa32Xaa33Xaa34Xaa35 (S12); Xaa36Xaa37Xaa38Xaa39Xaa40Xaa41Xaa42Xaa43 (S13).

Xaa1 = P or I;
Xaa2 = V, I or R;
Xaa3 = P, V or I;
Xaa4 = L, M or Y;
Xaa5 = S, E, N or A;
Xaa6 = L, I or M;
Xaa7 = V, T, I, M, K or R;
Xaa8 = M, Y or Q;
Xaa9 = V or I;
Xaa10 = M, Y, L, or E;
Xaa11 = S, N, or A;
Xaa12 = M, I, or L;
Xaa13 = M, I, K, or R;
Xaa14 = A, G, or S;
Xaa15 = D, L, F, N or I;
Xaa16 = I or V;
Xaa17 = P, V or I;
Xaa18 = V or A;
Xaa19 = S, G, A or E;
Xaa20 = L, M, I, Y or F;
Xaa21 = R, Y, K, M, I or V;
Xaa22 = S, A or G;
Xaa23 = P or V;
Xaa24 = L or Y;
Xaa25 = L, M, I, Y or F;
Xaa26 = R, T, Y, V or I;
Xaa27 = S, A or G;
Xaa28 = N or I;
Xaa29 = K, V, I or R;
Xaa30 = P, V or I;
Xaa31 = F, Y, L, M or A;
Xaa32 = S or E;
Xaa33 = M, I, L, Y or F;
Xaa34 = M, K, I or R;
Xaa35 = M or A;
Xaa36 = F, L, D, I or V;
Xaa37 = I, K, V or D;

Xaa38 = P or V;
Xaa39 = any amino acid;
Xaa40 = S or A;
Xaa41 = L, I, M, Y or F;
Xaa42 = R, K or Y;
Xaa43 = M or A; and
Xaa = any amino acid.
ACTIVITY - Cytostatic; Antiinflammatory; Antibacterial.
No biological data is given.
MECHANISM OF ACTION - Inhibitor of protease activity.
USE - M1 is useful for determining AAS motif for CS of (I), where (I) is a matrix metalloproteinase, or a proteolytic enzyme that mediates the pathogenesis of a pathogen. (I) is a lethal factor of B. anthracis, pla and YopJ proteases of Yersinia, and the smallpox H1L metalloprotease. (I) is selected from protease of pathogenic organisms, cathepsin family protease, tumor necrosis factor- alpha converting enzyme (TACE), calpains, caspases, beta -site amyloid precursor protein cleaving enzyme (BACE, beta -secretase), presenilins, membrane-type serine proteases, furin and other proprotein convertases, proteasome components and proteases affecting the blood clotting cascade. M2 is useful for determining AAS motif for a binding site of (I). M3 is useful for identifying protease inhibitors, where cleavable protease substrate is an intramolecularly-quenched fluorogenic peptide protease substrate comprising a protease cleavage motif sequence flanked by an fluorescent group and a fluorescence quenching moiety. PI1, PI2 and PI3 are useful in preparing a medicament. (All claimed). PI1, PI2, and PI3 are useful to treat diseases, including pathogenic infections, cancer, inflammatory diseases.
Dwg.0/4

L11 ANSWER 2 OF 11 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2002-351647 [38] WPIDS
DOC. NO. CPI: C2002-099844
TITLE: New B-lymphocyte stimulator binding polypeptide useful in detecting or isolating BLyS or BLyS-like polypeptide comprises a specified amino acid **sequence.**
DERWENT CLASS: B04 D16
INVENTOR(S): BELTZER, J P; FLEMING, T J; LADNER, R C; POTTER, M D
PATENT ASSIGNEE(S): (DYAX-N) DYAX CORP
COUNTRY COUNT: 96
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2002016412	A2	20020228	(200238)*	EN	269

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PH PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG UZ VN YU ZA ZW

AU 2001085066 A 20020304 (200247)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2002016412 A2		WO 2001-US25891	20010817
AU 2001085066 A		AU 2001-85066	20010817

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2001085066 A	Based on		WO 200216412

PRIORITY APPLN. INFO: US 2000-226489P 20000818
AN 2002-351647 [38] WPIDS
AB WO 200216412 A UPAB: 20030206
NOVELTY - A B-lymphocyte stimulator binding polypeptide (A) comprising one of 13 4-18 residue amino acid **sequences**, all given in the specification, is new.

DETAILED DESCRIPTION - A B-lymphocyte (BLyS) stimulator binding polypeptide (A) comprising Asp-Xaa-Leu-Thr (I); T1-T2-T3-Cys-T5-Phe-T7-Trp-Glu-Cys-T11-T12-T13 (II); S1-S2-S3-Cys-S5-S6-S7-S8-S9-S10-Cys-S12-S13-S14 (III); U1-U2-U3-Cys-U5-U6-U7-U8-U9-U10-U11-Cys-U13-U14-U15 (IV); Y1-Y2-Y3-Cys-Y5-Y6-Y7-Y8-Y9-Y10-Y11-Y12-Cys-Y14-Y15-Y16 (V); Z1-Z2-Z3-Cys-Z5-Z6-Z7-Z8-Z9-Z10-Z11-Z12-Z13-Z14-Cys-Z16-Z17-Z18 (VI); G1-G2-G3-G4-G5-G6-G7-G8-G9-G10-G11-G12 (VII); H1-H2-H3-H4-H5-H6-H7-H8-H9-H10-H11-H12 (VIII); Cys-K2-Phe-K4-Trp-Glu-Cys (IX);

Cys-J2-J3-J4-J5-J6-J7-Cys (X); Cys-M2-M3-M4-M5-M6-M7-M8-Cys (XI); Cys-N2-N3-N4-N5-N6-N7-N8-N9-Cys (XII); or Cys-Q2-Q3-Q4-Q5-Q6-Q7-Q8-Q9-Q10-Q11-Cys (XIII).

Xaa = Pro (P), Ser (S), Thr (T), Phe (F), Leu (L), Tyr (Y), Cys (C) or Ala (A);

T1 = A, Asn (N), Lys (K), or S;

T2 = A, Glu (E), Met (M), S, or Val (V);

T3 = A, N, K, or P;

T5 = F, Trp (W), or Y;

T7 = P or Y;

T11 = A, Gln (Q), His (H), F, or V;

T12 = N, Q, Gly (G), H, S, or V;

T13 = A, N, G, Ile (I), P, or S;

S1, S2, S3 = A, Asp (D), E, Q, G, H, I, L, K, M, F, P, S, T, W, Y, or V (S3may also be N);

S5 = D, I, L, or Y;

S6 = Arg (R), D, Q, H, I, L, K, F, P, Y, or V;

S7 = H, L, K, or F;

S8 = L, P, or T;

S9 = R, N, G, H, I, K, M, or W;

S10 = A, E, Q, G, H, I, L, M, F, S, W, Y, or V;

S12 = D, Q, E, G, I, L, K, F, S, W, Y, or V;

S13 = A, R, N, D, Q, E, G, H, L, K, M, F, P, S, T, W, Y, or V;

S14 = A, R, N, D, Q, E, G, H, I, L, K, F, P, W, Y, or V;

U1 = A, R, N, D, L, K, F, P, S, or T;

U2 = N, D, Q, H, I, K, P, T, or W;

U3 = A, R, N, E, Q, H, F, P, or T;

U5 = N, D, P, S, or T;

U6 = R, D, I, L, M, P, or V;

U7 = A, I, L , P, T, or V;

U8 = N, H, I, L, K, F, or T;

U9 = N, Q, G, H, L, K, M, P, or T;
U10 = R, N, D, Q, E, G, I, K, M, P, S, or T;
U11 = R, E, G, K, F, S, W, or Y;
U13 = Q, E, I, L, F, P, S, Y, or V;
U14 = N, G, I, F, P, T, W, or Y;
U15 = N, D, E, L, K, M, P, or T;
Y1 = N, D, H, L, F, P, S, or Y;
Y2 = R, N, D, H, F, S, or W;
Y3 = N, D, L, P, S, or V;
Y5 = D, Q, H, I, L, K, M, F, or T;
Y6 = H, I, L, M, F, P, T, or Y;
Y7 = D, H, L, or S;
Y8 = A, R, D, E, L, F, P, or T;
Y9 = A, R, N, or L;
Y10 = I, L, M, P, S, or T;
Y11 = A, R, N, G, H, K, S, or Y;
Y12 = A, R, N, Q, L, M, S, W, Y, or V;
Y14 = D, G, L, F, Y, or V;
Y15 = N, H, L, P or Y;
Y16 = N, D, H, F, S or Y;
Z1 = R, D, G, H, L, F, P, S, W, or Y;
Z2 = A, R, N, D, G, P, or S;
Z3 = R, N, Q, E, G, K, M, P, W or V;
Z5 = R, N, Q, E, H, L, F, P, W, Y, or V;
Z6 = R, D, Q, G, I, K, F, T, W or Y;
Z7 = A, R, D, E, G, L, S, or Y;
Z8 = D, Q, E, L, M, F, P, S, or Y;
Z9 = D, L, P, T, or V;
Z10 = R, Q, H, I, L, K, M, F, T, W or Y;
Z11 = A, R, N, Q, E, H, L, K, M, or T;
Z12 = A, N, Q, G, L, K, F, P, T, W, or Y;
Z13 = A, R, Q, H, K, M, F, P, T, W, or Y;
Z14 = R, Q, E, G, H, L, M, F, P, S, T, Y, or V;
Z16 = R, D, G, H, K, M, F, P, S, or W
Z17 = R, N, D, G, H, F, P, S, W or Y;
Z18 = A, R, N, D, H, L, F, or W;
G1 = A, R, G, H, L, K, M, F, W, Y, or V;
G2 = A, R, Q, H, I, L, F, T, W, or Y;
G3 = A, D, K, F, T, W or Y;
G4 = R, D, Q, K, M, F, P, S, Y, or V;
G5 = D, L, K, F, P, S, or V;
G6 = H, I, L, P, S, or T;
G7 = R, G, H, L, K, M, or T;
G8 = A, R, N, I, L, K, M, or T;
G9 = A, N, R, D, E, G, H, L, M, S, W, Y, or V;
G10 = I, L, F, S, T, W, Y, or V;
G11 = A, R, G, H, I, L, K, P, S, T, W, Y, or V;
G12 = R, D, H, L, K, M, F, P, S, W, Y, or V;
H1 = D, Q, E, G, H, K, M, or W;
H2 = R, Q, H, I, L, or P;
H3 = D, G, I, K, T, Y or V;
H4 = N, D, Q, E, M, P, S, or Y;
H5 = N, D, H, I, L, M, P, T or V;
H6 = D, E, H, L, K, P, or V;
H7 = R, N, Q, H, I, L, M, P, or T;
H8 = Q, G, H, L, M, S, or T;
H9 = N, Q, G, H, L, K, S, or T;
H10 = A, G, I, L, K, M, or F;

H11 = A, E, H, I, L, M, S, T, W, Y, or V;
H12 = R, Q, E, G, H, I, K, Y, or V;
K2 = F, W, or Y;
K4 = P or Y;
J2 = D, I, L, or Y;
J3 = R, D, E, H, I, L, K, F, P, Y, or V;
J4 = H, L, K, or F;
J5 = L, P, or T;
J6 = R, N, G, H, I, K, M or W;
J7 = A, N, Q, E, G, H, I, L, M, F, S, W, Y, or V;
M2 = N, D, P, S, or T;
M3 = R, D, I, L, M, P, or V;
M4 = A, I, L , P, T, or V;
M5 = N, H, I, L, K, F, or T;
M6 = N, E, G, H, L, K, M, P, or T;
M7 = R, N, D, Q, E, G, I, K, M, P, S, or W;
M8 = R, E, G, K, F, S, W, or Y;
N2 = D, Q, H, I, L, K, M, F, or T;
N3 = H, I, L, M, F, P, W, or Y;
N4 = D, H, L, or S;
N5 = A, R, D, E, L, F, P, or T;
N6 = A, R, N, or L;
N7 = I, L, M, P, S, or T;
N8 = A, R, N, G, H, K, S, or Y;
N9 = A, R, N, Q, L, M, S, W, Y, or V;
Q2 = R, N, Q, E, H, L, F, P, W, Y, or V;
Q3 = R, D, Q, G, I, K, F, T, W or Y;
Q4 = A, R, D, E, G, L, S, or Y;
Q5 = D, Q, E, L, M, F, P, S, or Y;
Q6 = D, L, P, T, or V;
Q7 = R, Q, H, I, L, K, M, F, T, W or Y;
Q8 = A, R, N, Q, E, H, L, K, M, or T;
Q9 = A, N, Q, G, L, K, F, P, T, W, or Y;
Q10 = A, R, Q, H, K, M, F, P, T, W, or Y;
Q11 = R, Q, E, G, H, L, M, F, P, S, T, Y, or V.

INDEPENDENT CLAIMS are also included for the following:

(1) a recombinant bacteriophage expressing exogenous DNA encoding (A);

(2) detecting BLyS or a BLyS-like polypeptide in a solution by contacting with (A) and determining binding;

(3) purifying BLyS or a BLyS-like polypeptide;

(4) a BLyS separation media comprising a chromatographic matrix material and an immobilized BLyS binding molecule (A);

(5) separating BLyS or a BLyS-like polypeptide from a solution using the medium of (d);

(6) a polynucleotide encoding (A);

(7) identifying a binding molecule for a BLyS target;

(8) a BLyS affinity maturation library comprising a population of at least 103 polypeptides containing amino acid sequence Ala-Xa1-Xa1-Xa1-Asp-Xa1-Leu-Thr-Xa1-Leu-Xa1-Xa1-Xa1-Xa1; and

(9) a DNA template encoding a multiplicity of (A) comprising a 42 nucleotide sequence, given in the specification.

Xa1 = any amino acid.

USE - In detection, isolation and/or purification of BLyS in a solution such as water or a buffer solution as well as any fluid and/or cell obtained from an individual biological fluid, body tissue, body cell, cell line, tissue culture or other source containing BLyS or BLyS-like polypeptides. The biological fluids

include sera, plasma, lymph, blood, blood fraction, urine, synovial fluid, spinal fluid, saliva or mucous.

ADVANTAGE - (A) binds reversibly or irreversibly BLyS and/or BLyS-like polypeptides with high affinity.

Dwg.0/0

L11 ANSWER 3 OF 11 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2002-329382 [36] WPIDS
DOC. NO. CPI: C2002-095090
TITLE: Novel compounds, useful for treating depressed neutrophil count, comprise peptide chains of approximately 6 to 40 amino acids in length that bind to granulocyte-colony stimulating factor receptor.
DERWENT CLASS: B04
INVENTOR(S): BALU, P; CWIRLA, S E; DUFFIN, D J; MCEOWEN-MERRILL, B; PIPLANI, S; SCHATZ, P J
PATENT ASSIGNEE(S): (GLAX) GLAXO GROUP LTD
COUNTRY COUNT: 95
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2002007676	A2	20020131	(200236)*	EN	90
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
AU 2001077073	A	20020205	(200236)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2002007676	A2	WO 2001-US23046	20010720
AU 2001077073	A	AU 2001-77073	20010720

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2001077073	A Based on	WO 200207676

PRIORITY APPLN. INFO: US 2000-620091 20000720
AN 2002-329382 [36] WPIDS
AB WO 200207676 A UPAB: 20020610
NOVELTY - Compounds (A) comprising a peptide chain approximately 6 to 40 amino acids in length that binds to granulocyte-colony stimulating factor receptor (G-CSFR) are new. The compounds contain specific **sequences** of amino acids (I-VII), all **sequences** are fully defined in the specification.

DETAILED DESCRIPTION - A compound comprising a peptide chain approximately 10 to 40 amino acids in length that binds to G-CSFR and contains a **sequence** of amino acids of formula CX1X2X3X4X5X6X7X8C (SEQ ID NO:1) (I), where each amino acid is

indicated by the standard one-letter abbreviation, is new.

X1 = A, N, S, F, D, G, L, T, E, V, P, Q, H, M or K;
X2 = M, G, R, H, D, I, V, A, S, E, N, F, Y, P, C, W or T;
X3 = E, V, W, F, M, A, N, S, L, T, Y, G or P;
X4 = V, I, G, Q, W, M, T, Y, L, P, D, C, E or A;
X5 = M, E, W, L, P, N, I, T, V, F, Y, Q, S., R, W, G, H, or D;
X6 = H, A, W, Y, V, F, Q, M, N, E, S, D, P or G;
X7 = M, F, Y, V, N, L, H, D, S, W, G, Q, C or T; and
X8 = C, Y, R, I, K, W, L, E, M, H, A, T, F, D, P, G or Q.

INDEPENDENT CLAIMS are also included for:

(1) a compound comprising a peptide chain approximately 9 to 40 amino acids in length that binds to G-CSFR and contains a **sequence** of amino acids of formula XI1XI2XI3SGWVWXI4 (SEQ ID NO:2) (II);

(2) a compound comprising a peptide chain approximately 6 to 40 amino acids in length that binds to G-CSFR and contains a **sequence** of amino acids of formula ERXII1XII2XII3C (SEQ ID NO: 3);

(3) a compound comprising a peptide chain approximately 9 to 40 amino acids in length that binds to G-CSFR and contains a **sequence** of amino acids of formula XIII1MVYXIII2XIII3PXIII4W (SEQ ID NO: 4) (IV);

(4) a compound comprising a peptide chain approximately 12 to 40 amino acids in length that binds to G-CSFR and contains a **sequence** of amino acids of formula CXIV1XIV2XIV3XIV4XIV5XIV6XIV7XIV8XIV9XIV10C (SEQ ID NO:5) (V);

(5) a compound comprising a peptide chain approximately 9 to 40 amino acids in length that binds to G-CSFR and contains a **sequence** of amino acids of formula XV1XV2XV3XV4XV5XV6XV7XV8 (SEQ ID NO: 6) (VI);

(6) a compound comprising a peptide chain approximately 10 to 40 amino acids in length that binds to G-CSFR and contains a **sequence** of amino acids of formula XVI1XVI2XVI3XVI4XVI5XVI6XVI7XVI8XVI9 (SEQ ID NO: 7) (VII);and

(7) a compound comprising a peptide chain approximately 6 to 40 amino acids in length that binds to G-CSFR and contains a **sequence** of amino acids consisting of SEQ ID NO: 433 to SEQ ID NO: 489.

Each amino acid is indicated by the standard one-letter abbreviation.

XI1 = S, Q, R, L or Y;
XI2 = N, S, T, A or D;
XI3 = E, D or N;
XI4 = L, V, T, P or H;
XII1 = D, L, S, G, E, A, K or Y;
XII2 = W, Y, F, L or V;
XII3 = F, G, M or L;
XIII1 = D or E;
XIII2 = A or T;
XIII3 = Y or V;
XIII4 = P or Y;
XIV1 = E, G, P, N, R, T, W, S, L, H, A, Q or Y;
XIV2 = S, T, E, A, D, G, W, P,L, N, V, Y, R or M;
XIV3 = R, Y, V, Q, E, T, L, P, S, K, M, A or W;
XIV4 = L, M, G, F, W, R, S, V, P, A, D, C, or T;
XIV5 = V, T, A, R, S, L, W, C, I, E, P, H, F, D or Q;
XIV6 = E, Y, G, T, Q, M, S, N, A or P;
XIV7 = C, V, D, G, L, W, E, V, I, S, M or A;

XIV8 = S, Y, A, W, P, V, L, Q, G, K, F, I, E, or D;
XIV9 = R, W, M, D, H, V, G, H, Q, L, S, E or Y;
XIV10 = M, L, I, S, V, P, W, F, T, Y, R or Q;
XV1 = E, C, Q, V or Y;
XV2 = E, A, L, M, S, W or Q;
XV3 = K, R or T;
XV4 = L, A or V;
XV5 = R, A, M, H, E, V, L, G, D, Q or S;
XV6 = E or V;
XV7 = A or G;
XV8 = R, H, G or L;
XVI1 = A, E or G;
XVI2 = E, H or D;
XVI3 = R or G;
XVI4 = K, Y, M, N, Q, R, D, I, S or E;
XVI5 = A, S or P;
XVI6 = E, D, T, Q, K or A;
XVI7 = R, W, K, L, S, A or Q;
XVI8 = R or E; and
XVI9 = W, G or R.

ACTIVITY - Immunostimulant.

MECHANISM OF ACTION - Granulocyte-colony stimulating factor (G-CSF) modulator; Granulocyte-colony stimulating factor receptor (G-CSFR) agonist.

The binding affinities of (A) to G-CSFR was determined by G-CSF radioligand binding assay. The assay was conducted over a range of peptide concentrations and the results were graphed such that the y-axis represented the amount of bound 125I labeled G-CSF and the x-axis represented the concentration of peptide or peptide mimetic. The concentration at which the peptide or peptide mimetic reduced by 50% (IC50) the amount of 125I labeled G-CSF bound to immobilized G-CSFR was determined; the peptides gave varied IC50 values of less than or equal to 200 micro M or at most 500 micro M.

USE - The compounds (A) are used for treating conditions associated with depressed neutrophil count e.g. chemotherapy-induced neutropenia, AIDS-induced neutropenia or community-acquired pneumonia-induced neutropenia (all claimed). The compounds are useful: in vitro as tools for understanding the biological role of granulocyte-colony stimulating factor (G-CSF), including evaluation of many factors thought to influence, and be influenced by, production of white blood cells; in the development of compounds that bind to G-CSFR; as reagents for detecting G-CSF receptor or related receptor on living cells, fixed cells, in biological fluid, in tissue homogenates or in purified natural biological materials; in situ staining, fluorescence-activated cell sorting (FACS), Western blotting or enzyme-linked immunoadsorptive assay (ELISA); in receptor purification or in purifying cells expressing G-CSFR on the cell surface (or inside permeabilized cells); as commercial research reagent for various medical and diagnostic uses; or to treat a disease that would benefit from the ability to of a compound to mimic the effects of G-CSF in vivo.

ADVANTAGE - The compounds bind specifically to G-CSFR and allow for studies of biological activities mediated by the receptor and for the treatment of diseases, disorders and conditions that would benefit from activating or inactivating G-CSFR.

Dwg.0/0

L11 ANSWER 4 OF 11 WPIDS (C) 2003 THOMSON DERWENT

ACCESSION NUMBER: 2003-066797 [06] WPIDS
DOC. NO. CPI: C2003-017323
TITLE: Novel isolated ATP binding cassette A9 (ABCA9) transporter polypeptide useful as target for developing modulating agents to regulate transport of neurotoxic molecules, e.g. beta-amyloid peptide, across blood-brain barrier.
DERWENT CLASS: B04 D16 P14
INVENTOR(S): CHEN, H; CONNOP, B P; LE BIHAN, S; NATHWANI, P S; NATHWANI, P
PATENT ASSIGNEE(S): (ACTI-N) ACTIVE PASS PHARM INC
COUNTRY COUNT: 100
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 2002123106	A1	20020905	(200306)*		46
WO 2002070692	A2	20020912	(200306)	EN	

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SD SE SG SI SK SL TJ TM TN TR TT TZ UA UG US UZ VN YU ZA ZM ZW

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
US 2002123106	A1 Provisional	US 2001-273618P	20010305
	Provisional	US 2001-309096P	20010731
	Provisional	US 2001-315687P	20010828
		US 2002-90454	20020304
WO 2002070692	A2	WO 2002-CA275	20020304

PRIORITY APPLN. INFO: US 2002-90454 20020304; US 2001-273618P 20010305; US 2001-309096P 20010731; US 2001-315687P 20010828

AN 2003-066797 [06] WPIDS

AB US2002123106 A UPAB: 20030124

NOVELTY - An isolated polypeptide which comprises:

(a) a fully defined ATP binding cassette A9 (ABCA9) transporter polypeptide **sequence** of 1624 amino acids (S2) as given in specification, or its functional fragment;

(b) a naturally occurring allelic variant of (S2) (the allelic variant binds to antibody that selectively binds to (S2));

(c) an amino acid **sequence** that is at least 90% similar to (S2); and/or

(d) a functional fragment of (b) or (c), is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for:

(1) an isolated nucleic acid molecule encoding an ABCA9 transporter or its functional fragment which:

(a) comprises a nucleotide **sequence** that is at least 90% identical to a portion of, or a fully defined full-length **sequence** of 5018 (S1) or 4875 (S3) nucleotides as given in

the specification;

(b) encodes a polypeptide comprising an amino acid **sequence** at least about 90% similar to a portion of, or the full-length **sequence** of (S2); or

(c) comprises a nucleotide **sequence** which hybridizes to the **sequence** of (S1) or (S3);

(2) an oligonucleotide primer comprising at least 12 contiguous nucleotides of (S1) or (S3) or its complement from a region specific to ABCA9 transporters;

(3) an isolated nucleic acid molecule comprising a nucleotide **sequence** which is complementary to (II);

(4) an isolated nucleic acid molecule comprising (II) and a nucleotide **sequence** encoding a heterologous polypeptide;

(5) a vector (III) comprising (II);

(6) a host cell (IV) transfected with (III);

(7) preparation of (I);

(8) an antibody (V) which selectively binds to (I);

(9) detecting (M1) presence of (II) in a sample involves contacting the sample with a nucleic acid probe or primer which selectively hybridizes to the nucleic acid molecule in the sample to thereby detect the presence of (II) in the sample;

(10) a kit comprising a compound which selectively binds to (I) or a compound which selectively hybridizes to (II), and instructions for use;

(11) modulating (M2) the activity of (I) involves contacting the polypeptide or a cell expressing a polypeptide with a compound which binds to the polypeptide to modulate the activity of the polypeptide;

(12) a composition comprising (II) having a **sequence** of (S1) or its functional fragment, and a carrier;

(13) a composition comprising an antisense oligonucleotide capable of specifically hybridizing to a portion, or the full-length, of (S1), and a carrier; and

(14) a transgenic knockout mouse whose genome comprises a homozygous disruption in its endogenous ABCA9 gene, where the homozygous disruption prevents the expression of a functional ABCA9 protein, an results in the transgenic knockout mouse being sterile.

ACTIVITY - Neuroprotective; Nootropic; Antiparkinsonian; Anticonvulsant.

MECHANISM OF ACTION - Modulator of amyloid beta -protein export; Modulator of human ABCA9 transporter activity; Gene therapy.

No biological data given.

USE - (I) Is useful for identifying a compound which binds to it and for identifying a compound which modulates its activity.

(II) Is useful for detecting allelic variation of (S1) or its ortholog in a biological sample which involves obtaining a polynucleotide that hybridizes to (S1) or its ortholog, from the sample; and determining whether the polynucleotide is identical to a portion or full-length of (S1) or its ortholog.

(IV) Is useful for producing (I) by recombinant techniques. (V) Is useful for detecting the presence of (I) in a sample (all claimed).

(I) Is useful as target for developing modulating agents to regulate a variety of cellular processes, particularly, the transport of neurotoxic molecules, e.g., beta -amyloid peptide (A beta) across cell membrane or the blood-brain barrier (BBB). (I) is useful as target for developing diagnostic carriers and therapeutic agents for detecting and/or treating cells or tissues having

multi-drug resistance, e.g., cancer.

(M2) is useful for treating Alzheimer's disease, prion diseases, Huntington's disease, Parkinson's disease, amyotrophic lateral sclerosis (ALS), spinocerebellar ataxia, frontotemporal dementia, etc.

(I) is useful as an immunogen to produce (V).

(II) Is useful as probes or primers for detecting ABCA9-encoding nucleic acids. The probes and primers are useful for identifying and/or cloning other ABCA9 transporter family members as well as ABCA9 transporter homologs from other species. The probes can be used to detect transcripts or genomic sequences encoding the same or homologous proteins, and a part of a diagnostic test kit for identifying cells or tissue which mis-express ABCA9 transporter protein. (II) Can be inserted into vectors, and used as gene therapy vectors. (II) is useful for expressing (I) via a recombinant expression vector in a host cell in gene therapy applications, to detect ABCA9 mRNA, or genetic alteration in ABCA9 gene and to modulate ABCA9 activity.

Portions or fragments of (II) are useful to:

(i) map their respective genes on a chromosome, e.g., to locate gene regions associated with genetic disease or to associate ABCA9 with a disease;

(ii) identify an individual from a minute biological sample (tissue typing), and

(iii) aid in forensic identification of a biological sample.

(II) Can be inserted into vectors and used as gene therapy vectors.

(I) and (II) allow the development of:

(a) strategies to assist in the delivery of drugs to the brain;

(b) therapeutic treatment for Alzheimer's disease;

(c) new treatments for mood and panic disorders;

(d) agents capable of ameliorating multi-drug resistance;

(e) new treatments for diseases involving cholesterol mis-regulation; and

(f) new treatments for inflammatory diseases.

(I), (II) and (V) are useful for:

(i) screening assays;

(ii) predictive medicine (e.g., diagnostic assays, prognostic assays, monitoring clinical trials, and pharmacogenetics); and

(iii) methods of treatment (e.g., therapeutic and prophylactic).

(V) Is useful for isolating (I), to detect (I), to evaluate the abundance and pattern of expression of the protein, to diagnostically monitor protein levels in tissue, as a part of a clinical testing procedure, e.g., to determine the efficacy of a given treatment regimen.

(V) is also useful for isolating ABCA9 proteins, regulating the bioavailability of ABCA9 proteins and modulating ABCA9 activity.

Dwg.0/4

L11 ANSWER 5 OF 11 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2001-611273 [70] WPIDS
DOC. NO. CPI: C2001-182571
TITLE: Novel compounds comprising specific amino acids within CCR5 (HIV 1 co-receptor) amino terminal domain including negatively charged and two sulfated tyrosine residues is useful for treating HIV infection in humans.

DERWENT CLASS: B04 D16
INVENTOR(S): DRAGIC, T; OLSON, W C
PATENT ASSIGNEE(S): (AARO-N) AARON DIAMOND AIDS RES CENT; (PROG-N) PROGENICS PHARM INC; (DRAG-I) DRAGIC T; (OLSO-I) OLSON W C
COUNTRY COUNT: 95
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001064710	A2	20010907	(200170)*	EN	163
AU 2001047254	A	20010912	(200204)		
US 2002068813	A1	20020606	(200241)		

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001064710	A2	WO 2001-US6699	20010228
AU 2001047254	A	AU 2001-47254	20010228
US 2002068813	A1 Provisional	US 2001-267231P	20010207
		US 2001-796202	20010228

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2001047254	A Based on	WO 200164710

PRIORITY APPLN. INFO: US 2001-267231P 20010207; US 2000-185667P 20000229; US 2000-205839P 20000519; US 2001-796202 20010228

AN 2001-611273 [70] WPIDS

AB WO 200164710 A UPAB: 20011129

NOVELTY - Compounds (peptides) (I) containing specific amino acids within CCR5 (HIV 1 co-receptor) amino terminal domain including negatively charged and tyrosine residues are new. At least two tyrosine residues in the compound are sulfated.

DETAILED DESCRIPTION - (I) comprises the following formula (F1) using standard amino acid single letter nomenclature (excluding variables): theta alpha YXXYXXXE beta lambda (F1). alpha = 0-9 amino acids, providing there are more than 2 amino acids, they are joined by peptide bonds in consecutive order and have a **sequence** identical to a fully defined **sequence** of 352 amino acids (S1) as given in the specification, beginning with I at position 9 and extending from there in the amino terminal direction; beta = 0-13 amino acids, providing there are more than 2 amino acids, they are joined by peptide bonds in consecutive order and have a **sequence** identical to (S1) beginning with P at position 19 and extending from there in the carboxy terminal direction; theta = amino acid group or an acetylated amino group; lambda = represents a

carboxyl group or an amidated carboxyl group; and

X = any amino acid.

All of alpha Y, D, X, X, Y, X, X, X, E and beta are joined by peptide bonds and at least two tyrosines in the compound are sulfated.

INDEPENDENT CLAIMS are also included for the following:

(1) a composition (II) comprising a compound of formula:

(a) theta alpha YDINYYTSE beta lambda (preferred form of (I)), where at least two residues of tyrosine are sulfated, theta , alpha , lambda and beta are as described above; and

(b) a detectable marker attached to (I);

(2) a composition which comprises a carrier and (I) effective to inhibit binding of HIV-1 to CCR5 receptor on the surface of CD4+ cell;

(3) a compound of formula (F2): Delta -(alpha YDINYYTSE beta lambda)n, where at least two residues of tyrosine are sulfated, alpha , lambda and beta are as described above;

(4) a compound of formula (F3): (theta alpha YDINYYTSE beta)n- Delta , where at least two residues of tyrosine are sulfated, theta , alpha , and beta are as described above;

(5) identifying an agent which inhibits binding of a CCR5 ligand to a CCR5 receptor involves (M1)-(M5):

(a) (M1) comprises:

(i) immobilizing (I) on a solid support (M1);

(ii) contacting the immobilized compound with detectable CCR5 ligand to saturate all binding sites and to form a complex;

(iii) removing any unbound CCR5 ligand;

(iv) contacting the complex with the agent; and

(v) detecting whether any CCR5 ligand is displaced from the complex, where displacement of detectable CCR5 ligand from the complex indicates that the agent binds to the compound;

(b) (M2) comprises:

(i) contacting (I) with detectable CCR5 ligand to saturate all binding sites to form a complex;

(ii) removing any unbound CCR5 ligand;

(iii) measuring the amount of CCR5 ligand which is bound to the compound in the complex;

(iv) contacting the complex with the agent so as to displace CCR5 ligand from the complex; and

(v) measuring the amount of CCR5 ligand bound to the compound with the amount of CCR5 ligand which is bound to the compound in the complex in the absence of the agent

(c) (M3) comprises:

(i) immobilizing (I) on a solid support;

(ii) contacting the immobilized compound from step (i) with the agent and detectable CCR5 ligand to form a complex;

(iii) removing any unbound ligand;

(iv) measuring the amount ligand which is bound to the immobilized compound;

(v) measuring the amount ligand which binds to the immobilized compound in the absence of the agent; and

(vi) comparing the amount of CCR5 ligand which is bound to the immobilized compound in step (v) with the amount measured in step (iv);

(d) (M4) comprises:

(i) contacting (I) with the agent and detectable CCR5 ligand to form a complex;

(ii) removing any unbound ligand;

(iii) measuring the amount of ligand which is bound to the compound;

(iv) measuring the amount of ligand which binds to the compound in the absence of the agent; and

(v) comparing the amount of CCR5 ligand which is bound to the compound in step (iii) with the amount measured in step (iv);

(e) (M5) comprises:

(i) immobilizing the compound on a solid support;

(ii) contacting the immobilized compound with the agent dissolved or suspended in a known vehicle and measuring the binding signal generated by such contact;

(iii) contacting the immobilized compound with the known vehicle in the absence of the compound and measuring the binding signal generated by such contact; and

(iv) comparing the binding signal measured in step (ii) with the binding signal measured in step (iii); and

(6) obtaining (M6) a composition which involves identifying a compound which inhibits binding of a CCR5 ligand to a CCR5 receptor according to any one of the above mentioned methods and admixing the compound so identified or a homolog or its derivative with a carrier.

The formulae (F2) and (F3) comprise the following:

n = an integer from 1 to 8; Delta = a polymer; and

- = 8 linkers which attach the structure in parentheses to Delta ACTIVITY - Anti-HIV.

No supporting data given.

MECHANISM OF ACTION - Inhibitor of HIV binding to CCR5 receptors on surface of CD4+ cells; vaccine.

The ability of different CCR5 N-terminal (Nt) peptides (Sulfated peptides, Phosphorylated peptides, and Sulfated and Scrambled Peptides) to inhibit HIV-1 entry into CD4+CCR5+CXCR4+ cells was tested using a luciferase-based single round of entry assay.

Only peptides S-10/14 (sulfated) and S-3/10/14 (phosphorylated) inhibited the entry of the R5 isolate HIV-1(JR-FL) by approximately 50% in HeLa-CD4+CCR5+ and U87MG-CD4+CCR5+, but the entry of the R5X4 isolate HIV-1(DH123) and HIV-1(Gun-1), or of the X4 isolate HIV-1(HxB2) was not inhibited.

USE - (I) is useful for preventing and inhibiting human immunodeficiency virus infection of a CD4+ cell present in a subject, preferably human, which also carries a CCR5 receptor on its surface, comprising administering (I) to a CD4+ cell to inhibit binding of HIV to the CCR5 receptor.

(I) is also useful for treating a subject whose CD4+ cells are infected with HIV, by inhibiting binding of HIV to the CCR5 receptor on the subject's CD4+ cells.

The methods may be carried out in a subject infected (therapeutic method), not infected with HIV (prophylactic method), or in a subject who is not infected with but has been exposed to HIV.

(I) is also useful for identifying an agent which inhibits binding of a CCR5 ligand to a CCR5 receptor (claimed).

Dwg.0/13

L11 ANSWER 6 OF 11 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2001-335563 [35] WPIDS
DOC. NO. NON-CPI: N2001-242247
DOC. NO. CPI: C2001-103631

TITLE: Novel isolated or recombinant alpha-interferon homologues useful for inhibiting growth of tumors or inhibiting replication of virus and for treating autoimmune diseases such as multiple sclerosis, rheumatoid arthritis.

DERWENT CLASS: B04 D16 T01

INVENTOR(S): CHEN, T; HEINRICHS, V; PATTEN, P A

PATENT ASSIGNEE(S): (MAXY-N) MAXYGEN INC

COUNTRY COUNT: 95

PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001025438	A2	20010412	(200135)*	EN	209

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TZ UG ZW

W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW

AU 2000080013 A 20010510 (200143)

EP 1238082 A2 20020911 (200267) EN

R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001025438	A2	WO 2000-US27781	20001006
AU 2000080013	A	AU 2000-80013	20001006
EP 1238082	A2	EP 2000-970665	20001006
		WO 2000-US27781	20001006

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2000080013	A	Based on	WO 200125438
EP 1238082	A2	Based on	WO 200125438

PRIORITY APPLN. INFO: US 1999-415183 19991007

AN 2001-335563 [35] WPIDS

AB WO 200125438 A UPAB: 20010625

NOVELTY - Isolated or recombinant nucleic acid (NA) (I) comprising polynucleotide (PN) **sequence** encoding alpha -interferon (IFN) homologue(II), is new.

DETAILED DESCRIPTION - Isolated or recombinant nucleic acid (NA) (I) comprising a PN **sequence** which is:

(i) one of 35 (S1) defined **sequences** of 498 nucleotides or their complements;

(ii) PN encoding one of 35 (S2) defined polypeptide (PP) of 166 amino acids (aa) or their complements;

(iii) PN which hybridizes to a PN of (i) or (ii);

(iv) PN comprising a fragment of (i), (ii) or (iii) encoding a PP with antiproliferative activity in human Daudi-cell line-based assay;

(v) one of 7 (S3) defined **sequences** of 498 nucleotides or their complements;

(vi) PN encoding one of 7 (S4) defined polypeptides of 166 aa or their complements;

(vii) PN which hybridizes to a PN of (v) or (vi); or

(viii) PN comprising a fragment of (v), (vi) or (vii) encoding a PP with antiviral activity in a murine cell line/EMCV-based assay;

(ix) PN encoding PP (A);

(x) PN encoding PP comprising at least 20 aa of any one of (S2) and one or more of aa residues Ala19, (Tyr or Gln)34, Gly37, Phe38, Lys71, Ala76, Tyr90, Ile132, Arg134, Phe152, Lys160 or Glu 166;

(xi) PN encoding PP comprising at least 155 aa of any one of (S2) and aa residues Lys160 and Glu166;

All **sequences** are given in the specification.

INDEPENDENT CLAIMS are also included for the following:

(1) vector comprising (I);

(2) composition produced by digesting (I) with a restriction endonuclease, RNAse or DNAse or incubating (I) in the presence of deoxyribonucleotide triphospates and a NA polymerase;

(3) isolated or recombinant PP (III) encoded by (I);

(4) PP (IV) comprising at least 100 contiguous aa of a protein encoded by (I);

(5) PP (V) which is specifically bound by a polyclonal antisera raised against antigen comprising a **sequence** of (S2) or (S4) or its fragment, where the antisera is subtracted with an IFN alpha -PP encoded by a NA corresponding to one or more of GenBank accession number (GAN); J00210(alpha -D), J00207(alpha -A), X02958(alpha -6), X02956(alpha -5), V00533(alpha -H), V00542(alpha -14), V00545(IFN-IB), X03125(alpha -8), X02957(alpha -16), V00540(alpha -21), X02955(alpha -4b), V00532(alpha -C), X02960(alpha -7), X02961(alpha -10 pseudogene), R0067 (Gx-1), I01614, I01787, I07821, M12350(alpha -F), M38289, V00549(alpha -2a) and I08313(alpha -con1);

(6) antibody or antisera produced by administering (III) or (IV) to a mammal, which specifically binds to PP with a **sequence** of (S2) or S4) but not a GAN as in (5);

(7) preparation of (III) and (IV);

(8) making (M1) a modified or recombinant NA involves recursively recombining (I) with a **sequence** of one or more additional NAs, each encoding an alpha -IFN or its aa subsequence;

(9) NA library (VI) produced by (M1);

(10) cell population comprising (VI);

(11) recombinant IFN alpha -homologue NA (VII) produced by (M1);

(12) cell comprising (I), vector of (1) or (VII);

(13) producing (M2) modified or recombinant IFN alpha -homologue NA by mutating (I);

(14) modified or recombinant IFN alpha -homologue NA produced by (M2);

(15) computer or computer readable medium comprising a data base containing a sequence record comprising one or more character strings corresponding to a NA or protein sequence (S1-4);

(16) integrated system comprising (15), and comprising user input interface allowing user to selectively view one or more sequence records;

(17) NA comprising unique subsequence in a NA of (S1) or (S3), where the subsequence is unique as compared to known IFN alpha -NA or a NA sequence which corresponds to a GAN as described above;

(18) a PP which comprises a unique subsequence in (S2) or (S4) where the unique subsequence is unique as compared to a known IFN PP or a sequence of PP encoded by a NA corresponding to any of a GAN has described above;

(19) target NA which hybridizes under stringent conditions to a unique coding oligonucleotide which encodes a unique subsequence in (S2) or (S4);

(20) NA produced by (M1); and

(21) IFN alpha -PP or its aa subsequence produced by (M1).

CDLPQTHSLGX11X12RAX15X16 LLX19QMX22RX24SX26FSCLKDRX34DFGX38PX40 EEFDX45X46X47FQX50X51QAIX55X56X57HEX60X61QQTFNX67FSTKX72SSX75X76WX78 X79X80LLX83KX85X86TX88LX90QQLNX95LEACVX101QX103VX105X106X107X108TPLM NX114 DX116ILAVX121KYX124QRITLTLX132EX134KYSPCX140 WEVVRAEIMRSFSFSTNLQKRLRRKE or its conservatively substituted variation (A)

X11 and X72 = N or D;
X12 = R, S or K;
X15 = L or M;
X16 and X101 = I, M or V;
X19 = A or G;
X22 = G or R;
X24 = I or T;
X26 = P or H;
X34 = H,Y or Q;
X38, X57, X67, X85 and X124 = F or L;
X40 and X47 = Q or R;
X45 = G or S;
X46 = N or H;
X50, X121 and X134 = K or R;
X51 and X76 = A or T;
X55 = S or F;
X56 and X75 = V or A;
X60 and X61 = M or I;
X78 and X83 = E or D;
X79 = Q or E;
X80 = S,R,T or N;
X86 = S or Y;
X88, X103 and X108 = E or G;
X90 = Y,H or N;
X95 = D,E or N;
X105 = G or W;
X106 = V or M;
X107 = E,G, or K;
X114 = V,E, or G;
X116 = S or P;
X132 = T,I, or M;
X140 = A or S.

ACTIVITY - Cytostatic; antiviral; immunosuppressive; antirheumatoid; antiarthritic; antidiabetic; dermatological; antiinflammatory.

MECHANISM OF ACTION - Gene therapy.

Balbc/c mice received subcutaneous doses of phosphate buffered saline (PBS), IFN- alpha homologue CH2.2, or CH2.3, murine IFN-alpha 4, or human IFN- alpha 2a in daily subcutaneous dose of 2, 10 or 50 micro g for four consecutive days. On day 2, mice were exposed to lethal intranasal dose of vesicular stomatitis virus (VSV). Mouse-optimized IFN- alpha homologue, CH2.2 and CH2.3 were as effective or more effective than native murine IFN micro -IFN- alpha

4 in protecting mice from VSV. At the concentration tested, human IFN- alpha 2a was nearly completely ineffective in protecting mice from the virus.

USE - (II) is used for inhibiting growth of tumor cells e.g. human carcinoma and leukemia cells. (II) is used for inhibiting replication of a RNA virus e.g. HIV or DNA virus e.g. hepatitis B virus. (II) is also useful for treating an autoimmune disorder such as multiple sclerosis, rheumatoid arthritis, type I diabetes, lupus erythematosus.

The computer system is useful to present information.

(I) is useful as therapeutic or prophylactic in in vivo or ex vivo treatment, in diagnostic methods, gene therapy, producing antisense molecules, as immunogens, for recombinantly producing (II), as substrates for further reactions e.g. shuffling reactions or mutation reactions to produce new and/or improved IFN- alpha homologues. The NA libraries are used for engineering NAs, proteins, pathways, cells and/or organisms with new and/or improved characteristics. (II) is also useful as an adjuvant for stimulating or augmenting an immune response related to an antigen.

ADVANTAGE - (II) have increased potency against cancer cells, reduced toxicity towards non-target cells and have lower side effects.

Dwg.0/5

L11 ANSWER 7 OF 11 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2000-328722 [28] WPIDS
CROSS REFERENCE: 2001-475714 [51]
DOC. NO. NON-CPI: N2000-247469
DOC. NO. CPI: C2000-099548
TITLE: Peptide derivatives of protein kinase alpha D regions which selectively modulate the activity of protein kinases.
DERWENT CLASS: B04 D16 S03
INVENTOR(S): BEN-SASSON, S A
PATENT ASSIGNEE(S): (CHIL-N) CHILDRENS MEDICAL CENT; (YISS) YISSUM RES & DEV CO; (CHIL-N) CHILDRENS MEDICAL CENT CORP; (YISS) YISSUM RES DEV CO HEBREW UNIV JERUSALEM
COUNTRY COUNT: 90
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2000018895	A1	20000406	(200028)*	EN	148
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ TZ UG ZW					
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG UZ VN YU ZA ZW					
AU 9960590	A	20000417	(200035)		
EP 1115847	A1	20010718	(200142)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI					
CN 1319133	A	20011024	(200213)		
JP 2002525382	W	20020813	(200267)		153
US 2002160478	A1	20021031	(200274)		

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
WO 2000018895	A1		WO 1999-US22106	19990924
AU 9960590	A		AU 1999-60590	19990924
EP 1115847	A1		EP 1999-969737	19990924
			WO 1999-US22106	19990924
CN 1319133	A		CN 1999-811271	19990924
JP 2002525382	W		WO 1999-US22106	19990924
			JP 2000-572342	19990924
US 2002160478	A1	Cont of	US 1998-161094	19980925
			US 2002-38612	20020108

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9960590	A	Based on	WO 200018895
EP 1115847	A1	Based on	WO 200018895
JP 2002525382	W	Based on	WO 200018895

PRIORITY APPLN. INFO: US 1998-161094 19980925; US 2002-38612 20020108

AN 2000-328722 [28] WPIDS

CR 2001-475714 [51]

AB WO 200018895 A UPAB: 20021125

NOVELTY - A peptide derivative (A) of the protein kinase alpha D region comprising 5-30 amino acids and which modulates activity of the protein kinase, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) a peptide having one of 76 amino acid **sequences**, all fully defined in the specification, any one amino acid in the peptide can vary, being any natural amino acid or analog of them;

(2) peptides comprising (I)-(V) ,or a subsequence comprising at least 5 amino acids of (I)-(XII)

(a) (AA)1-23 (I);
(b) (BB)1-20 (II);
(c) (CC)1-21 (III);
(d) (DD)1-39 (IV);
(e) (EE)1-34 (V);
(f) (FF)1-20 (VI);
(g) (GG)1-31 (VII);
(h) (HH)1-18 (VIII);
(i) (II)1-18 (IX);
(j) (JJ)1-34 (X);
(k) (KK)1-21 (XI); and
(l) (LL)1-22 (XII);

(3) a method of identifying a peptide which modulates the activity of a protein kinase, comprising incubating a test compound comprising (A), with cells having cellular activities controlled by a protein kinase, and assessing the activity of the kinase in cells grown in the presence or absence of the peptide;

(4) a method of modulating the activity of a protein kinase in a subject, comprising administering (A);

(5) a method of detecting a ligand that binds to the alpha D region of a protein kinase, comprising providing (A), incubating the derivative with a sample to be tested, and detecting any

ligand-derivative binding pair formed during the incubation;

(6) an antibody that immunologically binds to the alpha D region of a protein kinase; and

(7) a method of producing antibodies that bind to the alpha D region of a protein kinase, comprising providing (A), and producing antibodies to the derivative.

AA1, 3, 4, 9, 13, BB4, 8, 12, 20, CC4, 9, 10, 13, EE1, 9, 13, 34, FF1, 4, 9, 13, 20, GG1, 3, 4, 9, 13, 16, 31, HH9, 13, 17, 18, II9, 13, 18, JJ4, 9, 13, 22, 23, 34, KK4, 9, 12, 13, 20, 21, LL1, 9, 13, 22 = V, I, L, M;

AA2 = D, T, E, S;

AA5, BB6, EE8, GG22, 23, JJ10, KK11, 18, LL14, 18 = Q, N;

AA6, 7, 22, BB19, CC7, DD7, EE4, 7, 15, 20, FF7, GG5, 7, 20, 25, HH2, 7, II7, JJ7, 17, 20, 26, KK7, 16, LL6, 7, 21 = A, G;

AA8, BB2, 7, 13, CC2, DD2, EE2, 11, 22, FF2, FF11, GG2, 8, 19, 21. HH11, II5, 11, JJ2, 8, 19, KK15, LL2, 8, 17 = D, E;

AA10, DD14, EE29 = H, R, K;

AA11, 23, BB1, 11, CC12, DD3, 12, EE12, FF3, 12, GG12, HH3, 12, II3, 10, 12, JJ1, 3, 12, KK3, 6, 10, 19, LL3 = Y, F, W;

AA12, GG6, HH4, II4, JJ6, 16, KK2, 14 = H;

AA14 = S, Y, T, F, W;

AA15, FF16, HH16 = Q, N, H;

AA16 = H, V, L, M, I;

AA17, EE32, HH5, JJ25 = G, D, E, A;

AA18 = V, E, N, Q, I, L, M, D;

AA19 = F, D, P, A, W, Y, E, G;

AA20 = N, G, Q, A;

AA21, LL16 = P, F, W, Y;

BB3 = F, H, W, Y;

BB5, II2 = H, D, E;

BB9, 10, HH14 = K, R, T, S;

BB14 = A, K, R, G;

BB15 = V, S, A, I, L, M, T;

BB16, JJ32 = A, P, G;

BB17 = L, P, E, I, M, V, D;

BB18 = T, P, S;

CC1, DD1, 36 = T, M, S, I, L, V;

CC3 = F, H, W, Y;

CC5 = S, N, C, A, E, T, Q, D, G;

CC6 = K, H, N, R, Q;

CC8, HH8, LL5 = S, N, T, Q;

CC11, DD8, EE26, JJ27 = D, N, E, Q;

CC14, DD17, EE6, 10, 16, 17, FF10, 15, 19, GG10, 14, 17, 26, HH6, JJ5, 11, 14, LL10 = K, R;

CC15 = G, E, D, N, S, T, Q, A;

CC16 = E, G, P, D, R, K, A;

CC17 = T, S, D, E, G, A;

CC18 = G, R, K, A;

CC19 = K, R, Q, G, S, I, A, N, T, L, M, V;

CC20 = Y, A, D, K, V, L, F, W, E, R, I, M, G;

CC21 = L, V, Q, I, M, N;

DD4 = A, C, S, T, G;

DD5 = G, R, F, K, W, Y;

DD6 = Y, H, F, W;

DD10 = L, V, S, I, M, T;

DD11 = D, N, T, E, Q, S;

DD15 = K, R, S, A, G, T;

DD16 = S, N, K, T, Q, R;

DD18 = V, H, D, N, I, L, M, E, Q;
DD19 = L, T, S, A, E, I, M, V, D, G;
DD20 = E, F, D, W, Y;
DD21 = T, L, F, S, V, I, M, W, Y;
DD22 = D, Q, S, L, P, E, N, T, I, M, V;
DD23 = P, H, N, C, Y, Q, F, W, S;
DD24 = A, H, K, R, G;
DD25 = F, S, P, D, E, W, Y, T;
DD26 = A, D, E, K, R, G;
DD27 = R, I, K, A, S, G, L, M, V, T;
DD28 = E, A, R, P, L, D, K, I, M, V, G;
DD29 = H, N, R, K, E, Q, D, or
DD30 = G, S, P, K, M, Q, F, T, R, I, L, V, N, W, Y, A;
DD31 = T, P, E, R, S, D, K;
DD32 = S, A, D, K, R, G, T, E;
DD33 = T, E, I, K, F, S, D, L, M, V, R, W, Y;
DD34 = L, F, E, R, D, I, M, V, W, Y, K;
DD35 = Y, G, K, A, F, W, R;
DD37 = N, E, V, G, Q, D, I, L, M, A;
DD38, JJ31 = A, P, E, D, G;
DD39, JJ24 = L, A, G, I, M, V;
EE3 = Y, C, F, W, S;
EE5, JJ15 = S, A, T, G;
EE14, II14 = Q, R, N, K;
EE18, 19, FF5, GG18, 24, 27, 28, 29, JJ18, KK5 = P;
EE21 = L, M, P, I, V;
EE23, LL12 = Y, L, F, W, I, M, V;
EE24 = C, S, W;
EE25 = Y, F, P, W;
EE27 = P, I, T, G, L, M, V, S, A;
EE28 = S, N, C, P, T, Q;
EE30 = N, V, P, S, Q, I, L, M, T;
EE31 = P, S, T;
EE33, FF14 = Q, P, N;
FF6 = S, Y, T, F, W, L, I;
FF8 = S, C, T;
FF17 = K, R, S, T;
FF18 = N, E, A, Q, D, G, I, L G;
GG11, 15, 30, HH1, II1, 8, 17, KK1, 8, 17 = S, T;
HH10 = Q, S, T;
II6 = H, M, N, I, L, V, Q;
II15 = R, L, C, S, K, I, M, V, T;
II16 = Q, T, A, Y, N, S, F, W, G;
JJ21 = K, V, M, R, I, L;
JJ28 = D, P, E;
JJ29 = V, P, R, I, L, M, K;
JJ30 = A, T, Q, S, N, G;
JJ33 = L, E, I, M, V, D;
LL4, 19, 20 = C, S;
LL11 = K, N;
LL15 = K, Q, R, N;
AA2, 8, 17-19, BB2, 5, 7, 13, 17, CC2, 5, 11, 15-17, 20, DD2, 8, 11, 18-20, 25, 26, 28, 29, 30-34, 37, 38, EE2, 11, 20, 22, 33 , FF2, 11, 18, GG2, 8, 19, 21, HH5, 11, II2, 5, 11, JJ8, 19, 25, 27, 28, 31, 33 , KK15, LL2, 8, 17 = an aliphatic, substituted aliphatic, benzyl, substituted benzyl, aromatic, or substituted aromatic ester or D or E;
ACTIVITY - Cytostatic; anti-diabetic; anorectic;

antiinflammatory; dermatological; immunosuppressive; immunomodulator; osteopathic; cardiant; vasotropic; antiarteriosclerotic.

MECHANISM OF ACTION - Protein Kinase Modulator.

USE - The peptides can be used as test peptides to identify protein kinase modulators. They can also be used to modulate the activity of a protein kinase in a subject, and in a method of detecting a ligand that binds to the alpha D region of a protein kinase. They may be used to produce antibodies that bind to the alpha D region of a protein kinase. All claimed. The peptides are useful in the treatment of diseases caused by over- or under-activity of a protein kinase, e.g. cancer, diseases caused by proliferation of smooth muscle (e.g. restenosis and atherosclerosis), skin disorders, diabetes, obesity, diseases of the central nervous system, inflammatory disorders, autoimmune diseases and other immune disorders, osteoporosis and cardiovascular diseases.

Dwg.0/6

L11 ANSWER 8 OF 11 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 1997-401885 [37] WPIDS
DOC. NO. CPI: C1997-129614
TITLE: Peptide(s) that bind to interleukin-5- receptor - for treatment of asthma, inflammatory skin conditions, osteoarthritis etc..
DERWENT CLASS: B04
INVENTOR(S): BARRETT, R W; CHEN, M; ENGLAND, B P; SCHATZ, P J; SLOAN, D; SLOAN, D D
PATENT ASSIGNEE(S): (AFFY-N) AFFYMAX TECHNOLOGIES NV; (GLAX) GLAXO GROUP LTD
COUNTRY COUNT: 78
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 5654276	A	19970805	(199737)*		42
WO 9857980	A1	19981223	(199906)#	EN	
RW: AT BE CH DE DK EA ES FI FR GB GH GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG ZW					
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH HU IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK TJ TM TR TT UA UG US UZ VN YU ZW					
AU 9734497	A	19990104	(199921)#		
NO 9906209	A	20000215	(200020)#		
EP 991659	A1	20000412	(200023)#	EN	
R: AL AT BE CH DE DK ES FI FR GB GR IE IT LI LT LU LV MC NL PT RO SE SI					
JP 2002512631	W	20020423	(200243)#		120
AU 753958	B	20021031	(200282)#		
NZ 501664	A	20021220	(200309)#		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
US 5654276	A	US 1995-478312	19950607
WO 9857980	A1	WO 1997-GB1618	19970616

AU 9734497	A	AU 1997-34497	19970616
NO 9906209	A	WO 1997-GB1618	19970616
		NO 1999-6209	19991215
EP 991659	A1	EP 1997-930613	19970616
		WO 1997-GB1618	19970616
JP 2002512631	W	WO 1997-GB1618	19970616
		JP 1999-503919	19970616
AU 753958	B	AU 1997-34497	19970616
NZ 501664	A	NZ 1997-501664	19970616
		WO 1997-GB1618	19970616

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9734497	A	Based on	WO 9857980
EP 991659	A1	Based on	WO 9857980
JP 2002512631	W	Based on	WO 9857980
AU 753958	B	Previous Publ.	AU 9734497
		Based on	WO 9857980
NZ 501664	A	Based on	WO 9857980

PRIORITY APPLN. INFO: US 1995-478312 19950607; WO 1997-GB1618 19970616; AU 1997-34497 19970616; NO 1999-6209 19991215; EP 1997-930613 19970616; JP 1999-503919 19970616; NZ 1997-501664 19970616

AN 1997-401885 [37] WPIDS

AB US 5654276 A UPAB: 19970915

New compound that binds to interleukin-5 receptor comprises; (a) a core **sequence** of amino acids of formula (I), its dimers and oligomers; having

CX1RX2X7X8X3X4X5WX6C (I)

X1 = D, **E**, **I**, **S**, T, W or Y;

X2 = D, F, G, I, L, S, V, W or Y;

X3 = D, E, G, L, N, S, T or W;

X4 = H or R;

X5 = A, K, R, S, T, V or W;

X6 = D, E, F, L, M, P, Q or V;

X7 = I or V, and

X8 = A or R (i) molecule wt. < 5000 Da, and (ii) a binding affinity to I15-R and expressed by a IC50 of at most 100 mu m, where (i) from 0-all of the CONH linkages of the peptide have been replaced by a linkage selceted from a CH2OCONR linkage, a phosphonate linkage, a CH2SO2NR linkage, a CH2NR linkage and a CONR6 linkage and a NHCONH linkage where

R = H or lower alkyl and

R6 = lower alkyl; where the N-terminus of the peptide or its mimetic is selected from NRR1, NRCOR, NRCOOr, NRsO2R NHCONHR, succinimide, benzyloxycarbonyl-NH or berryloxycarbonyl-NH having 1-3 substituents on the phenyl ring selected from lower alkyl, lower alkoxy, chloro and bromo, where R,R1 = H or lower alkl and the C-terminus has formula COR2; R2 = OH, lower alkoxyor NR3R4 and R3,R4 = H or lower alkyl, where the N of the NR-3R4 group can optinally be the amine group of the N-terminus of the peptide so as to form a cyclic peptide. Also claimed is an aerosol device comprising (I) in an carrier solution at 0.1-30 mg/ml and a means for convering the solution or dry powder into an aerosol form suitable for inhalalation.

USE - (I) can be used for the treatment of an inflammatory disroders of the respiratory tract, specifically asthma (claimed). (I) can also be used for the treatment of immunomediated inflammatory skin conditions such as urticardi and angioedema, eczematous dermatitis, and hyperproliferative skin disease such as psorasis. Also for rheumatoid arthritis, rheumatoid spondylitis, osteoarturitis, gouty, arthisit, inflammatory bowel disease and peptic ulcer.

ADVANTAGE - (I) have economical production greater chemical stability, enhanced pharmacological properties (e.g. half-life, absorption, potency and efficacy), altered specificity (e.g. a broad spectrum of biological activities) and reduced antigenicity.
Dwg.0/3

L11 ANSWER 9 OF 11 SCISEARCH COPYRIGHT 2003 ISI (R)
ACCESSION NUMBER: 96:777549 SCISEARCH
THE GENUINE ARTICLE: VN346
TITLE: THE RATES OF CONVERGENCE OF M-ESTIMATORS FOR PARTLY LINEAR-MODELS IN DEPENDENT CASES
AUTHOR: SHI P D (Reprint); CHEN X R
CORPORATE SOURCE: BEIJING UNIV, DEPT PROBABIL & STAT, BEIJING 100871, PEOPLES R CHINA; UNIV SCI & TECHNOL CHINA, GRAD SCH, BEIJING 100039, PEOPLES R CHINA
COUNTRY OF AUTHOR: PEOPLES REPUBLIC OF CHINA
SOURCE: CHINESE ANNALS OF MATHEMATICS SERIES B, (JUN 1996) Vol. 17, No. 3, pp. 301-316.
ISSN: 0252-9599.
DOCUMENT TYPE: Article; Journal
LANGUAGE: ENGLISH
REFERENCE COUNT: 32

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Consider the partly Linear model Y-i = X(i)'beta(0)+g(0)(T-i)+e(i), where {((Ti, X(i))}(infinity)(1) is a strictly stationary **sequence** of random variables, the **e(i)'s** are i.i.d. random errors, the Y-i's are real-valued responses, beta(0) is a d-vector of parameters, X(i) is a d-vector of explanatory variables, T-i is another explanatory variable ranging over a nondegenerate compact interval. Based on a segment of observations (T-1,X(1)',Y-1),...,(T-n,X(n)',Yn), this article investigates the rates of convergence of the M-estimators for beta(0) and g(0) obtained from the minimization problem

Sigma(i=1)(n) rho(Y-i-X(i)'beta-g(n)(T-i)) = min (beta is an element of R?d ,gn is an element of Fn')

where F-n is a space of B-spline functions of order m + 1 and rho(.) is a function chosen suitably. Under some regularity conditions, it is shown that the estimator of g(0) achieves the optimal global rate of convergence of estimators for nonparametric regression, and the estimator of beta(0) is asymptotically normal. The M-estimators here include regression quantile estimators, L(1)-estimators, L(p)-norm estimators, Huber's type M-estimators and usual least squares estimators. Applications of the asymptotic theory to testing the hypothesis H-0 : A'beta(0) = <(beta)over tilde> are also discussed, where <(beta)over tilde> is a given vector and A is a known d x d(0) matrix with rank d(0).

L11 ANSWER 10 OF 11 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 95116551 MEDLINE
DOCUMENT NUMBER: 95116551 PubMed ID: 7816842

TITLE: A test of lattice protein folding algorithms.
AUTHOR: Yue K; Fiebig K M; Thomas P D; Chan H S; Shakhnovich E I; Dill K A
CORPORATE SOURCE: Department of Pharmaceutical Chemistry, University of California, San Francisco 94143-1204.
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1995 Jan 3) 92 (1) 325-9.
Journal code: 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199502
ENTRY DATE: Entered STN: 19950217
Last Updated on STN: 19950217
Entered Medline: 19950209

AB We report a blind test of lattice-model-based search strategies for finding global minima of model protein chains. One of us (**E .I.S.**) selected 10 compact conformations of 48-mer chains on the three-dimensional cubic lattice and used their inverse folding algorithm to design HP (H, hydrophobic; P, polar) **sequences** that should fold to those "target" structures. The **sequences**, but not the structures, were sent to the UCSF group (K.Y., K.M.F., P.D.T., H.S.C., and K.A.D.), who used two methods to attempt to find the globally optimal conformations: "hydrophobic zippers" and a constraint-based hydrophobic core construction (CHCC) method. The CHCC method found global minima in all cases, and the hydrophobic zippers method found global minima in some cases, in minutes to hours on workstations. In 9 out of 10 **sequences**, the CHCC method found lower energy conformations than the 48-mers were designed to fold to. Thus the search strategies succeed for the HP model but the design strategy does not. For every **sequence** the global energy minimum was found to have multiple degeneracy with 10(3) to 10(6) conformations. We discuss the implications of these results for (i) searching conformational spaces of simple models of proteins and (ii) how these simple models relate to proteins.

L11 ANSWER 11 OF 11 SCISEARCH COPYRIGHT 2003 ISI (R)
ACCESSION NUMBER: 94:26668 SCISEARCH
THE GENUINE ARTICLE: MP044
TITLE: CHARACTERIZATION AND REDOX PROPERTIES OF HIGH-MOLECULAR-MASS CYTOCHROME-C(3) (HMC) ISOLATED FROM DESULFOVIBRIO-VULGARIS MIYAZAKI
AUTHOR: OGATA M; KIUCHI N; YAGI T (Reprint)
CORPORATE SOURCE: SHIZUOKA UNIV, DEPT CHEM, EDUC BLDG, OYA, SHIZUOKA 422, JAPAN (Reprint); SHIZUOKA UNIV, DEPT CHEM, EDUC BLDG, OYA, SHIZUOKA 422, JAPAN
COUNTRY OF AUTHOR: JAPAN
SOURCE: BIOCHIMIE, (1993) Vol. 75, No. 11, pp. 977-983.
ISSN: 0300-9084.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 23
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Summary - Two kinds of high molecular mass cytochrome c3 (Hmc)

were isolated from Desulfovibrio vulgaris Miyazaki, one from the membrane (mHmc), the other from the soluble fraction (sHmc). Molecular masses of both Hmcs determined by SDS-PAGE are 65 kDa. Each Hmc molecule is composed of a single polypeptide chain and contains 16 hemes. sHmc and mHmc have identical UV-visible visible absorption spectra of a typical c-type cytochrome (three peaks at 530, 419, and 355 nm in the ferri form, and four peaks at 553, 523, 419, and 325 nm in the ferro form), and similar amino acid composition. There is no peak at 695 nm in the ferri form. but a Soret peak (423 nm) with a shoulder at 432 nm, indicative of the presence of a high-spin heme, appeared at low reduction stage. Hmcs are fragile proteins and readily denatured by bubbling with gas or by stirring. From the redox titration of sHmc monitored spectroscopically in the presence of standard redox dyes, the midpoint potentials of 16 hemes (E1 approximately E16) were determined. These hemes are classified into four groups based on their **E**(**i**)**s**: positive (E1 = 60, E2 = 15 mV), slightly negative (E3 = E4 = E5 = E6 = E7 = -120, E8 = -125, E9 = -135 mV), negative (E10 = E11 = -190, E12 = E13 = -195, E14 = 205 mV), and very negative (E15 = E16 = -260 mV) groups (may be deviated from the true microscopic values due to heme-heme interactions). The role of Hmc in the energy metabolism of this bacterium is discussed.

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